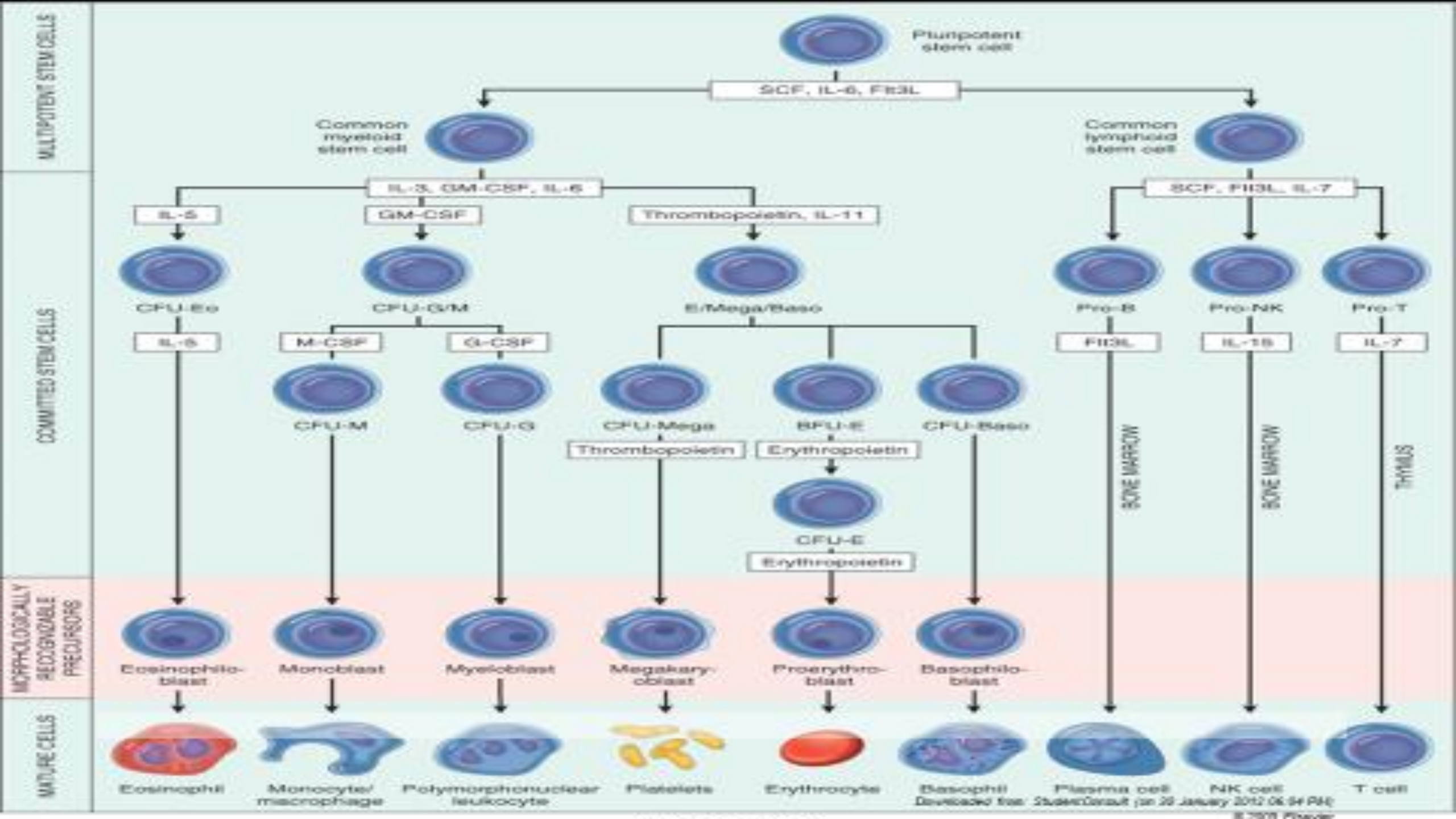
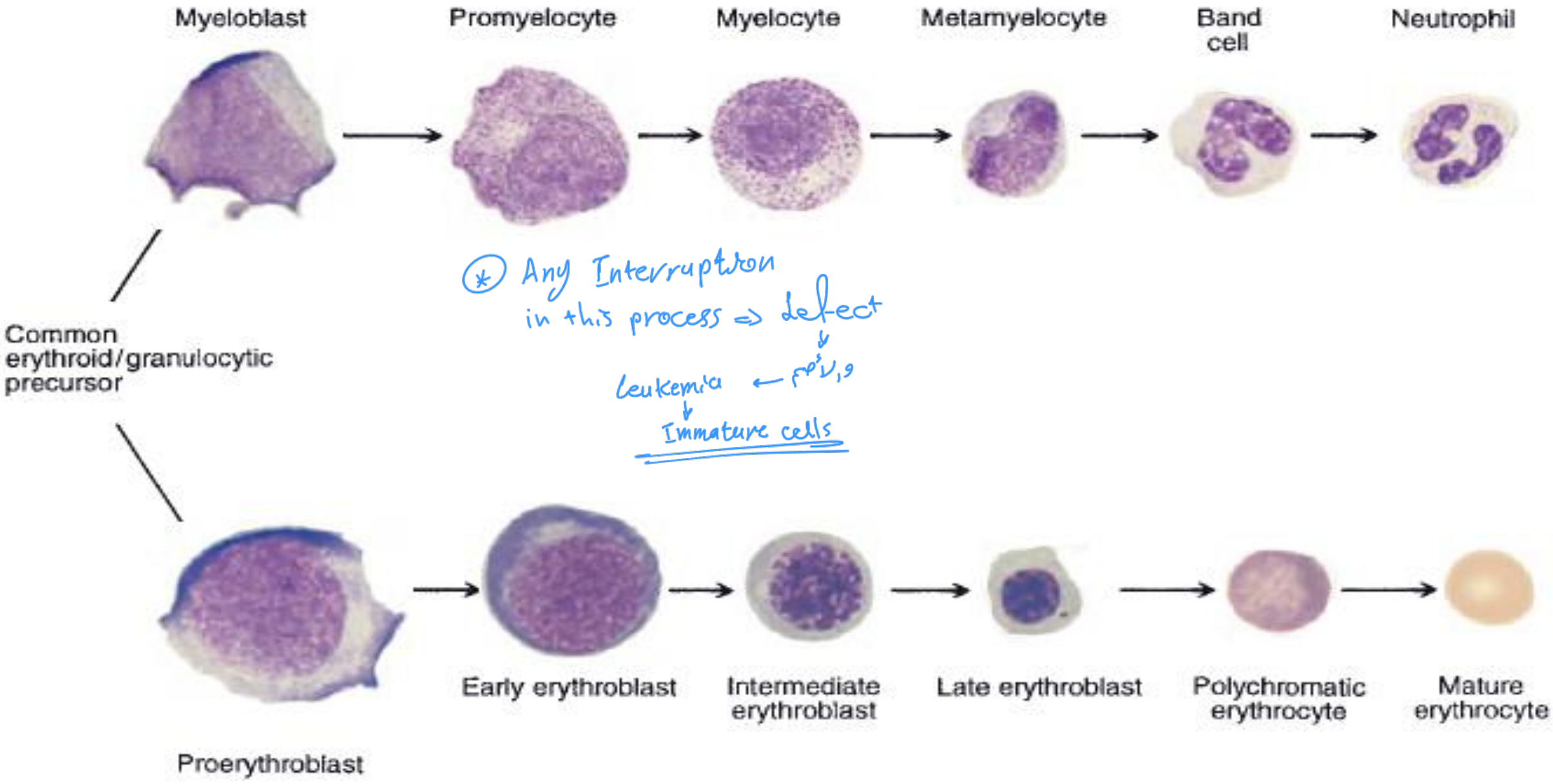
Hematology Lab

Sura Al Rawabdeh, MD

April 2022

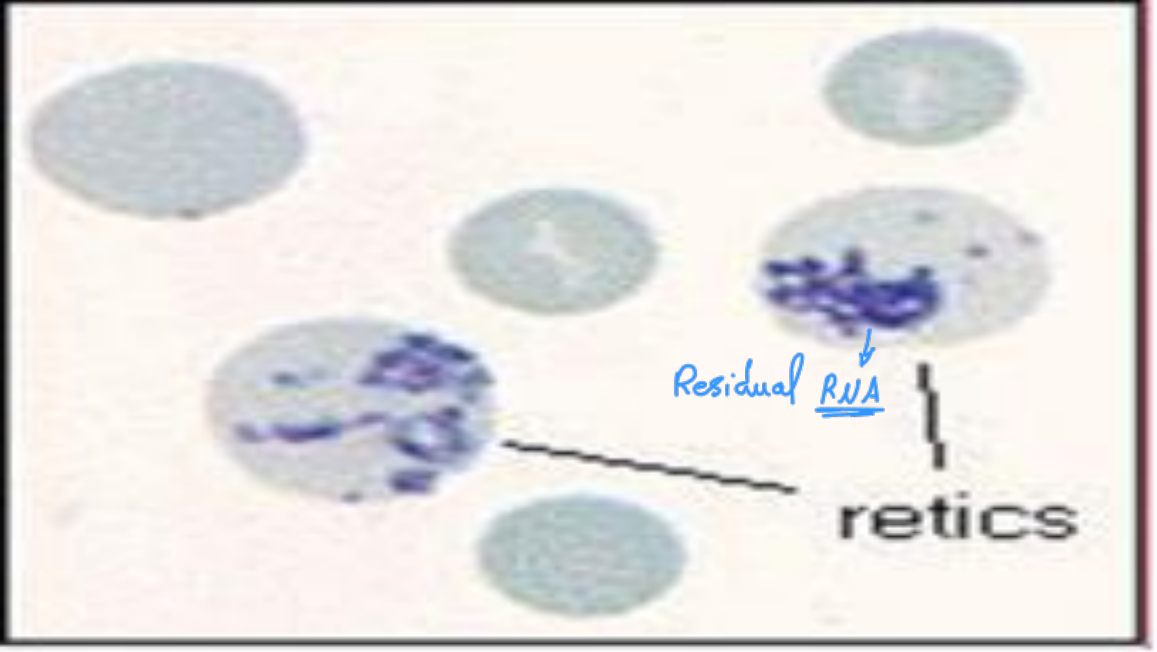


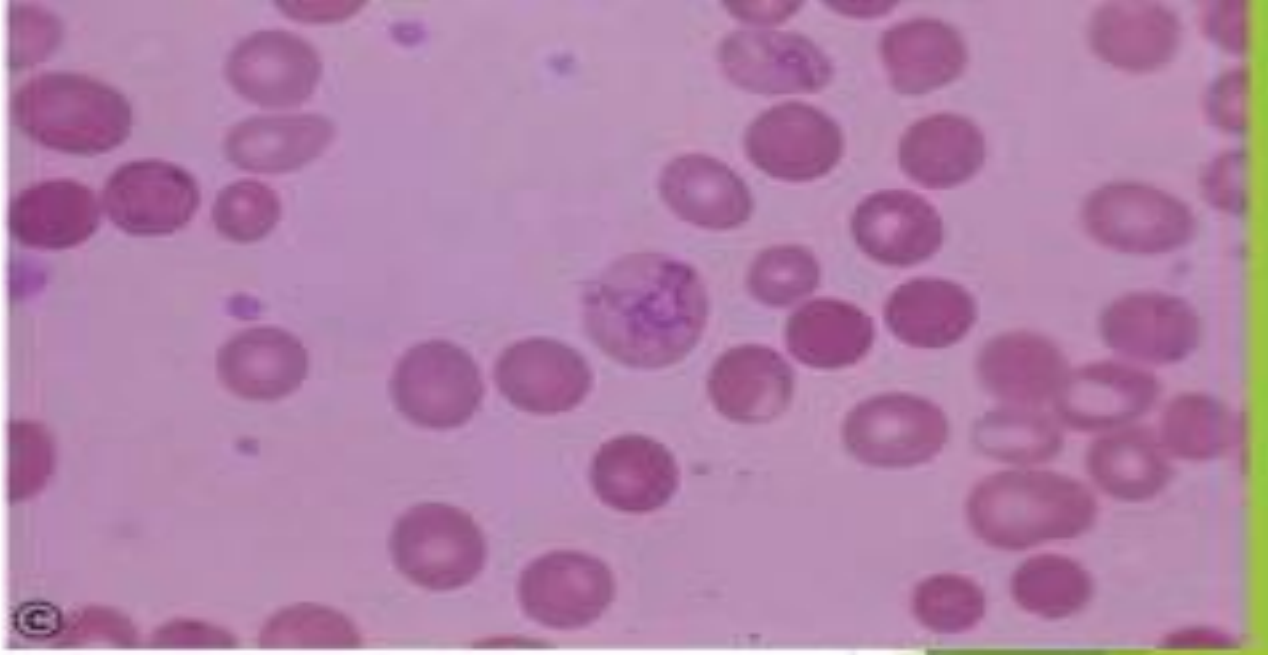


(*) Any Interruption in this process ⇒ defect

Leukemia ← Mutations

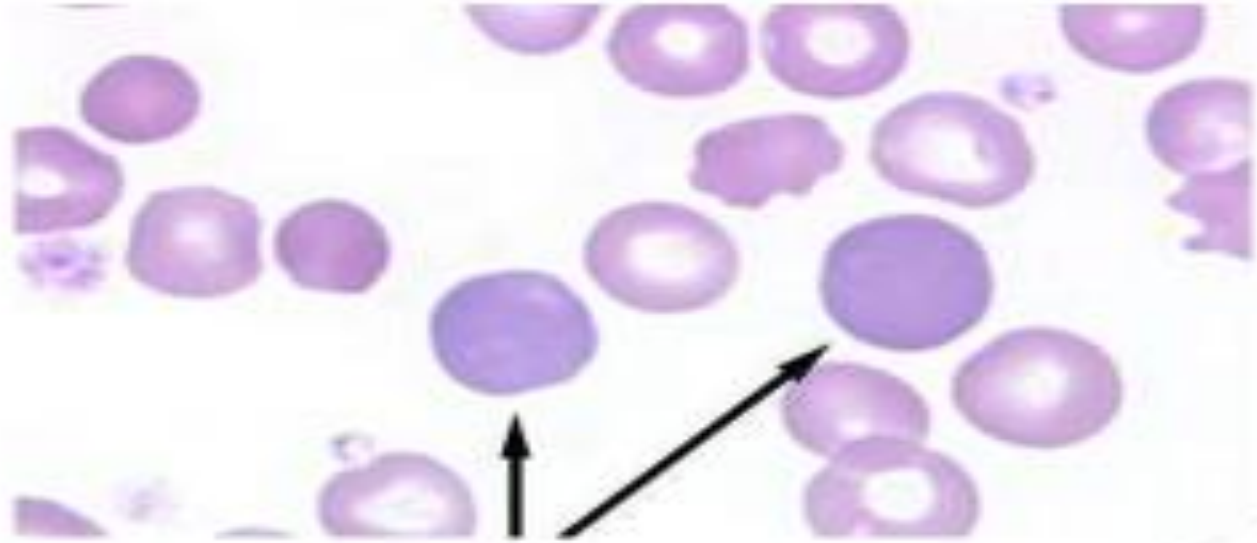
Immature cells

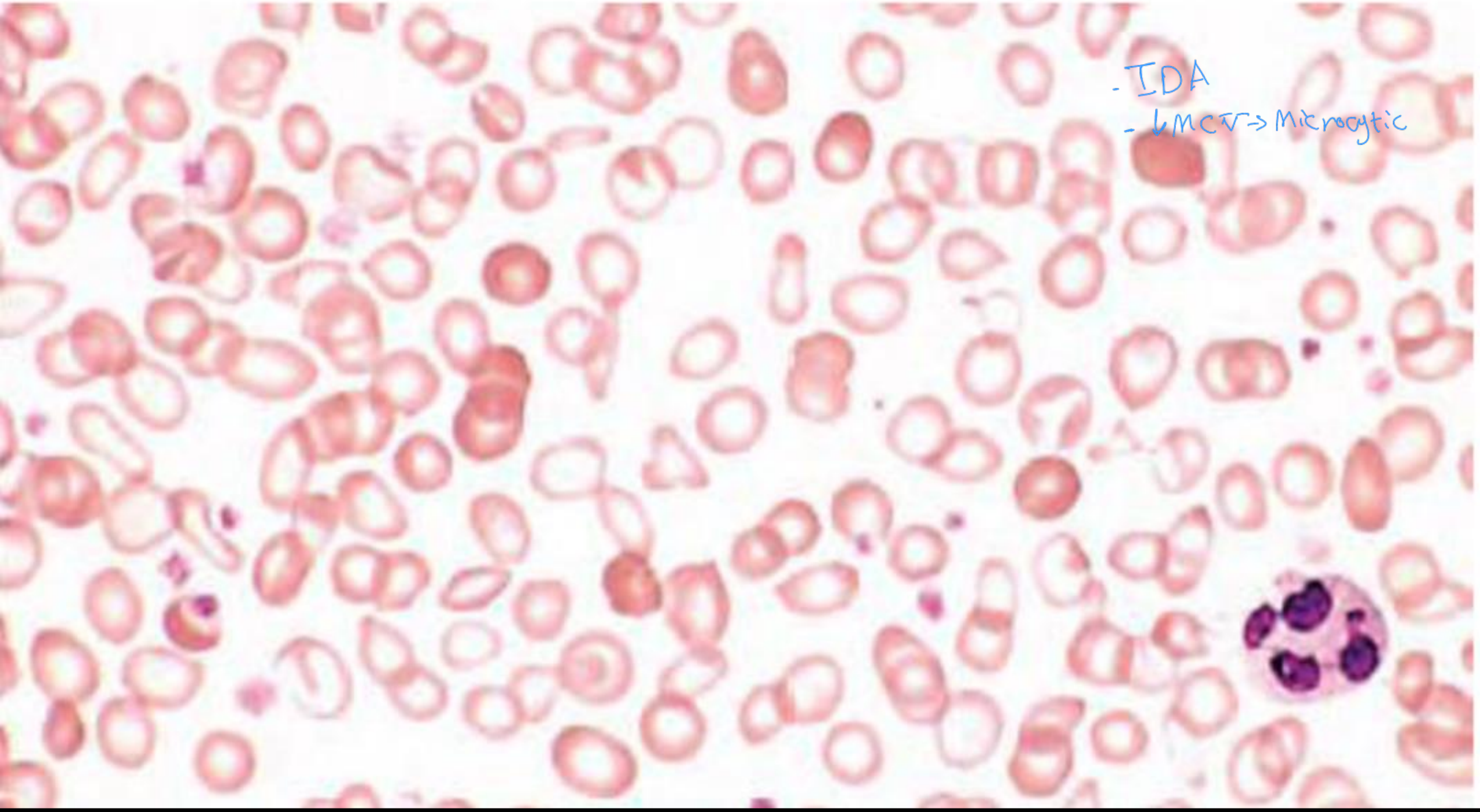


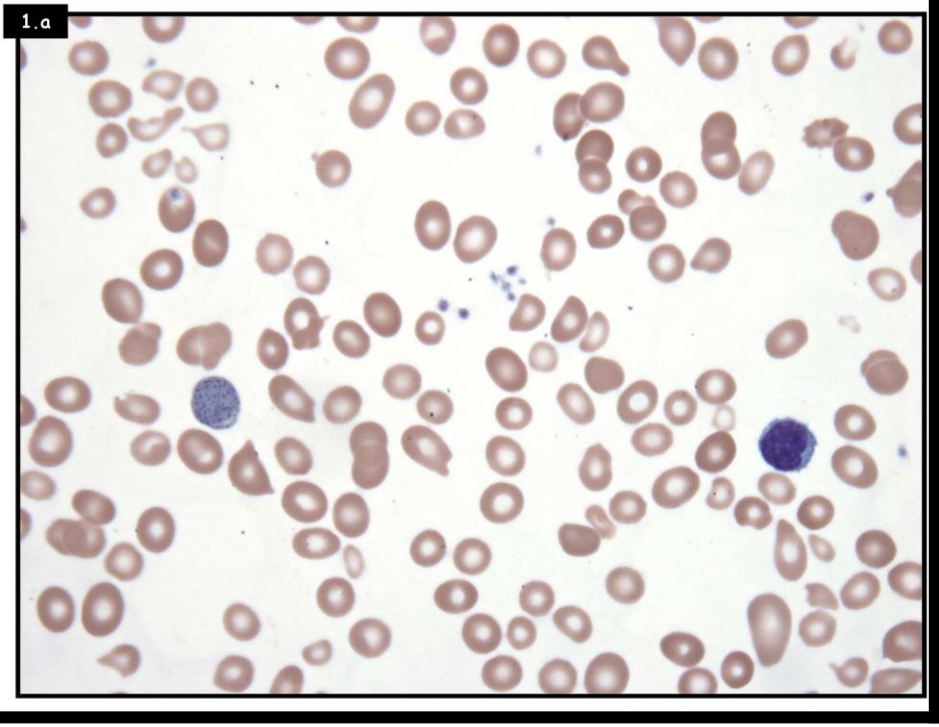


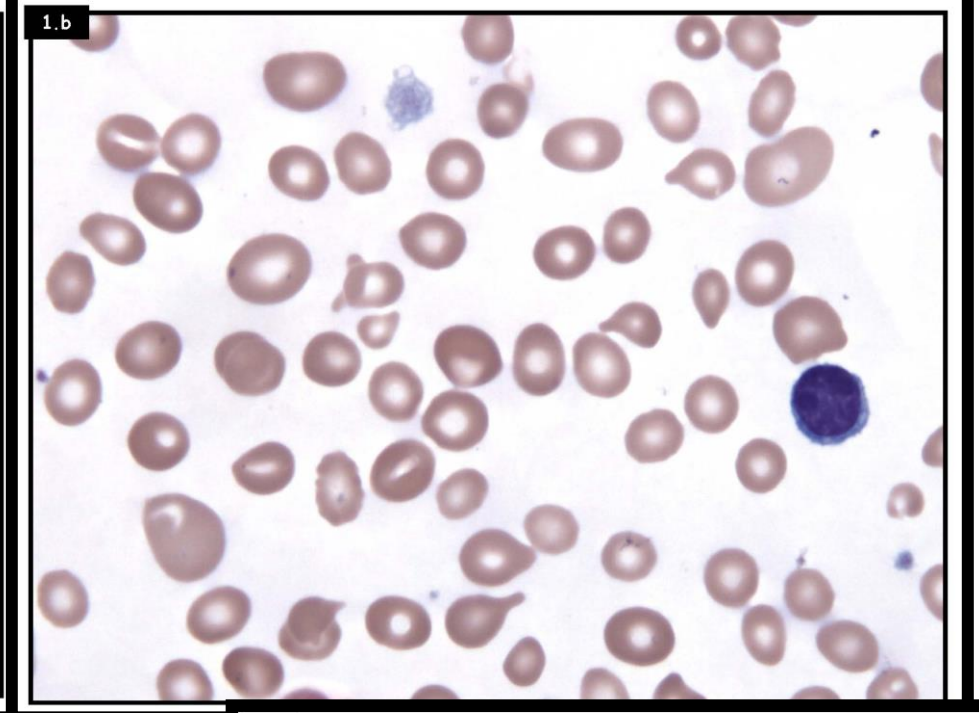
Anemia → good BM → Reticulo-cytosis

Anemia → del-ectel BM → ↓ retic count




- IDA
- ↓MCV -> Microcytic





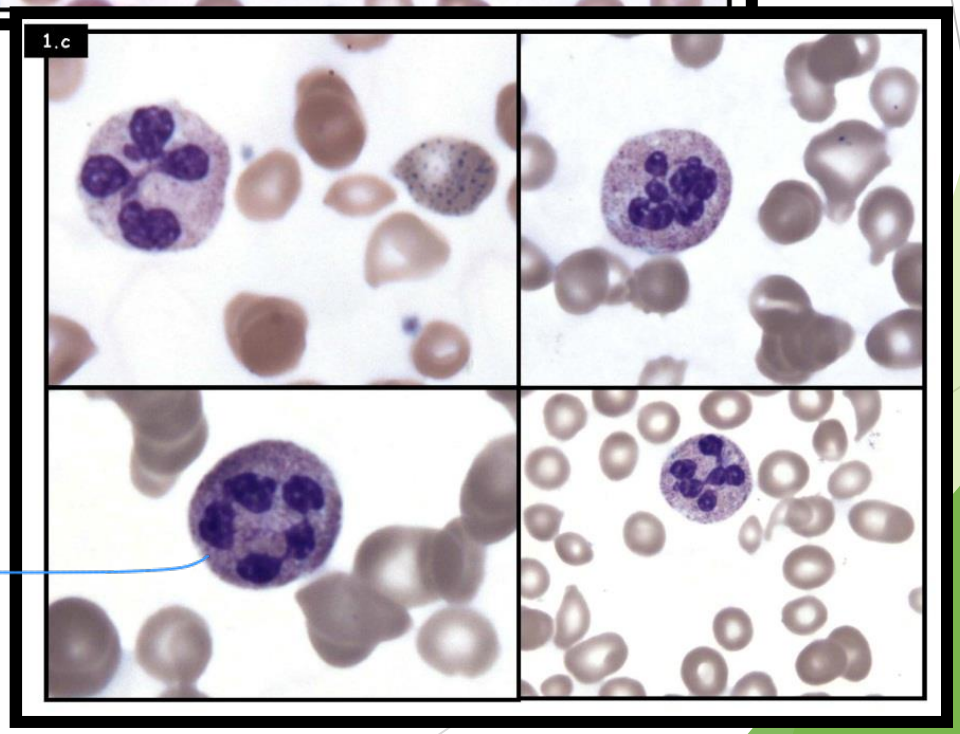
- ↑ MCV → Macrocytic
- Folate or B12 def.

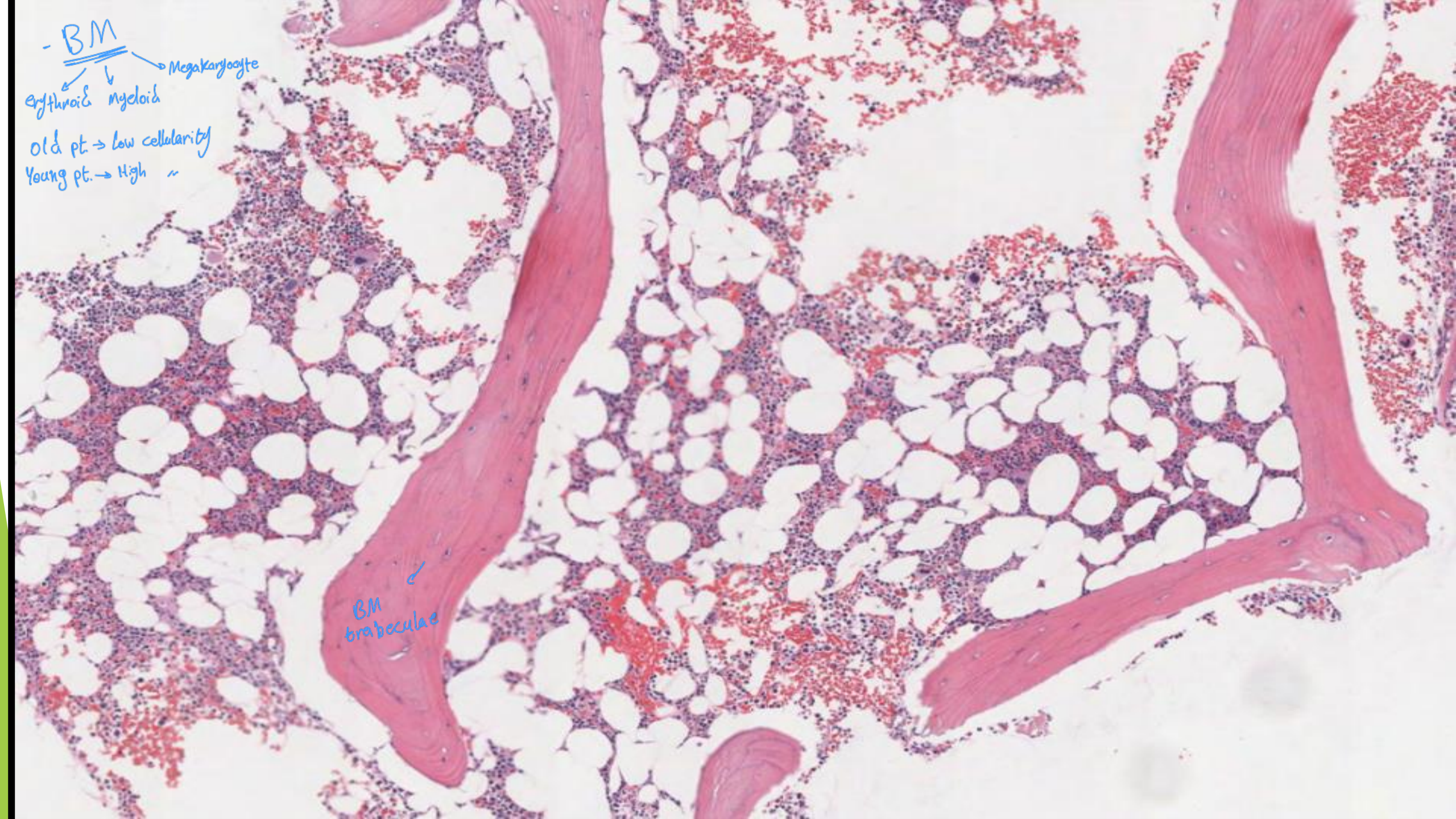
Macro-ovalocytes

different shapes + sizes of RBC's.

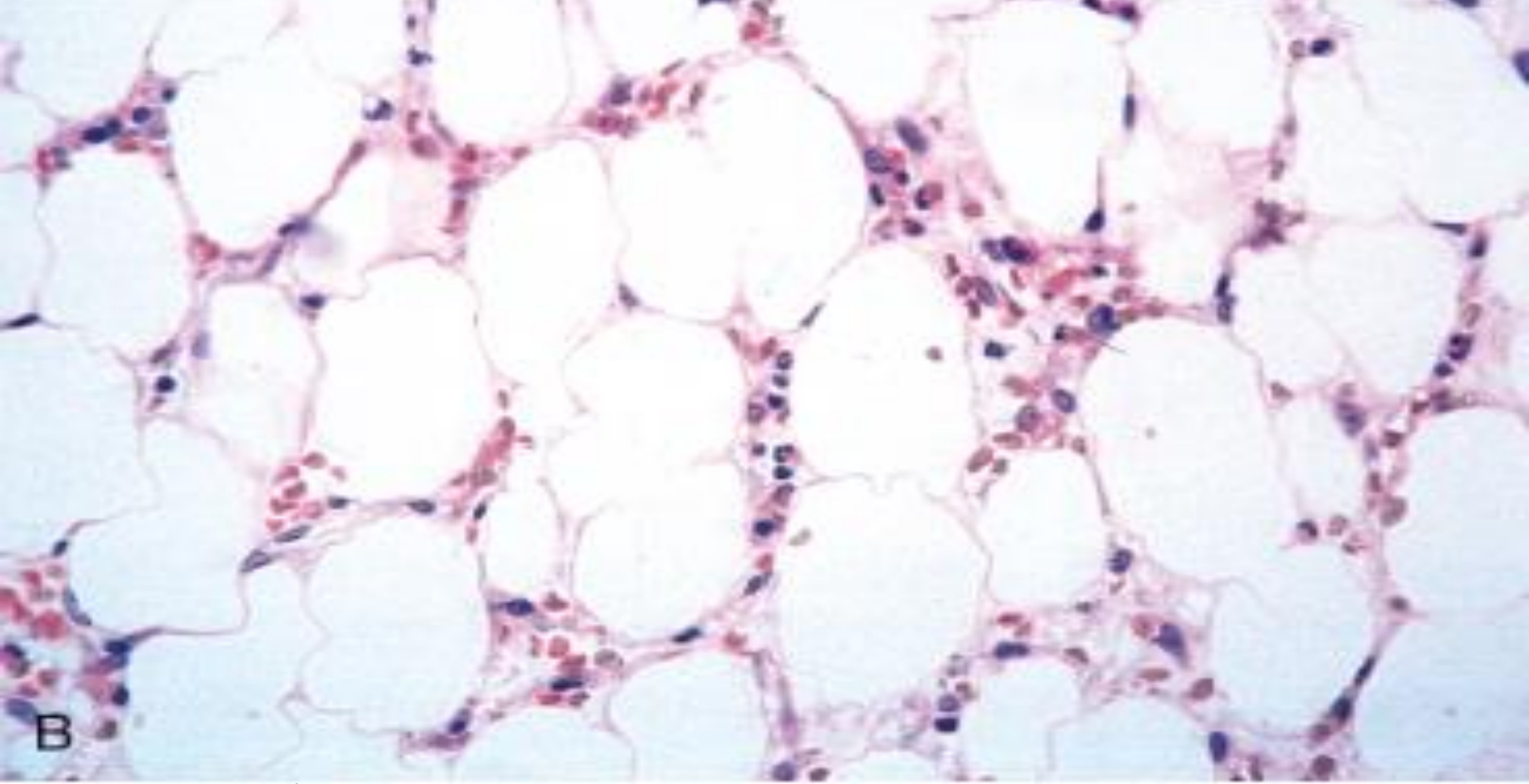
Moderate anisopoikilocytosis

Neutrophilic hypersegmentation

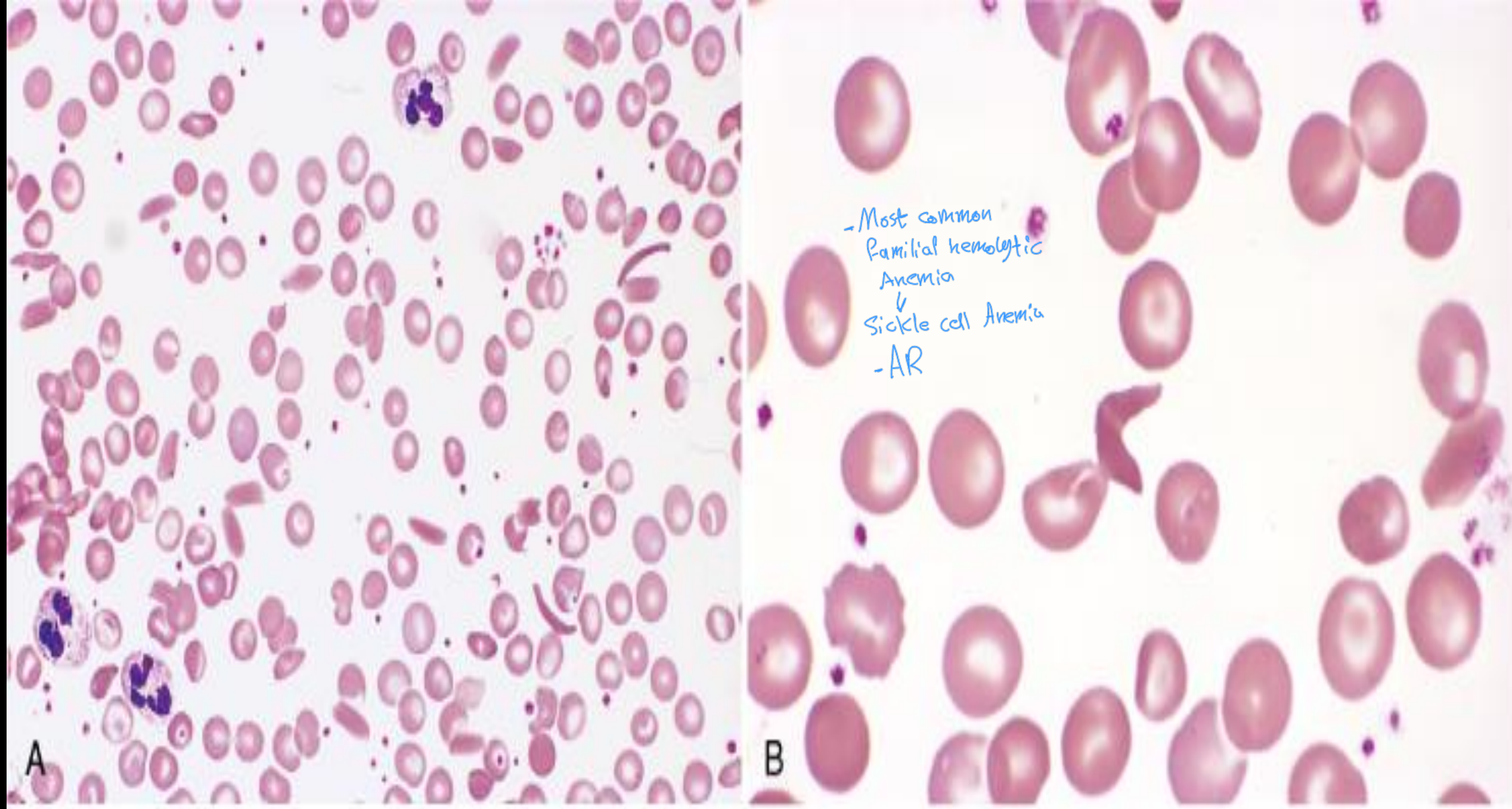



- BM
Megakaryocyte
erythroid
myeloid
old pt. → low cellularity
Young pt. → High
BM trabeculae


- Hypocellular
- Excess Fat
- Aplastic Anemia
Due: - Idiopathic
- Drugs
- Infections
Gr: ParvoVirus-B19 in G6PD
Hereditary spherocytosis
pathy.med.nagoya-u.ac



Aplastic Anemia



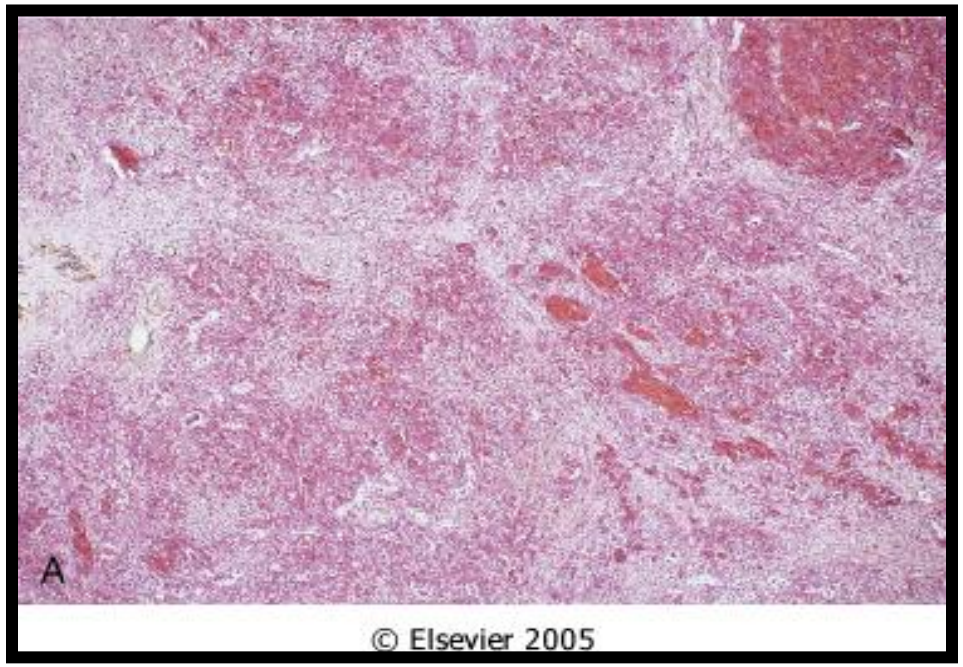
- Most common Familial hemolytic Anemia
  ↓
  Sickle cell Anemia
- AR

↳ sub of Glutamic acid → Valine at pos. 6 of AA.

Spleen in Sickle Cell Anemia

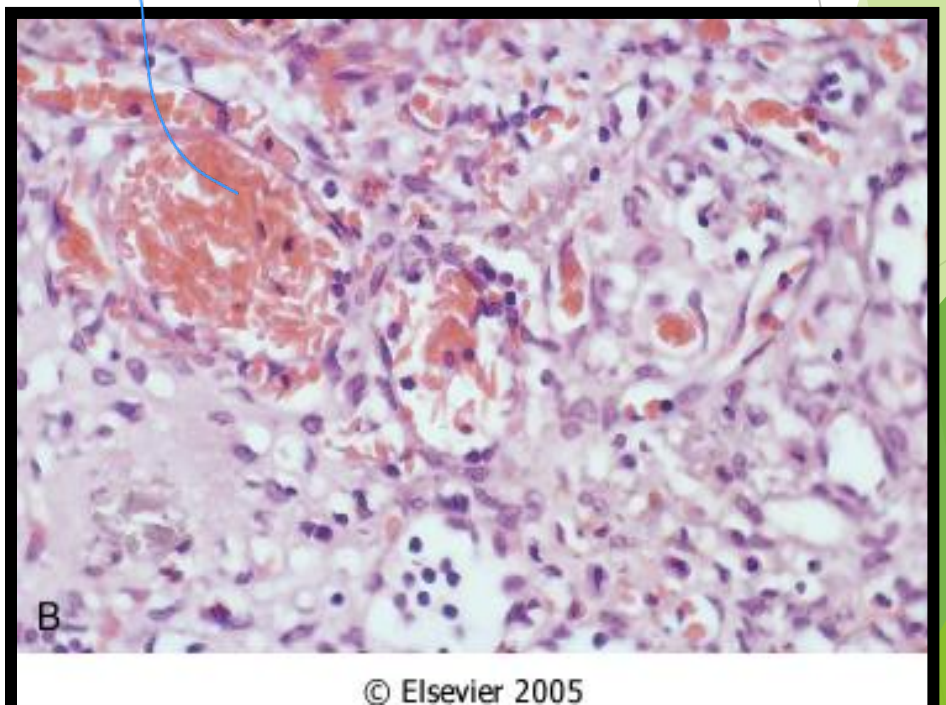
Red pulp ↳ obstructed

complication → also: - sluggish flow
- chronic hemolytic anemia → 20d life span.
↳ Micro-infarcts



RBC's sickled

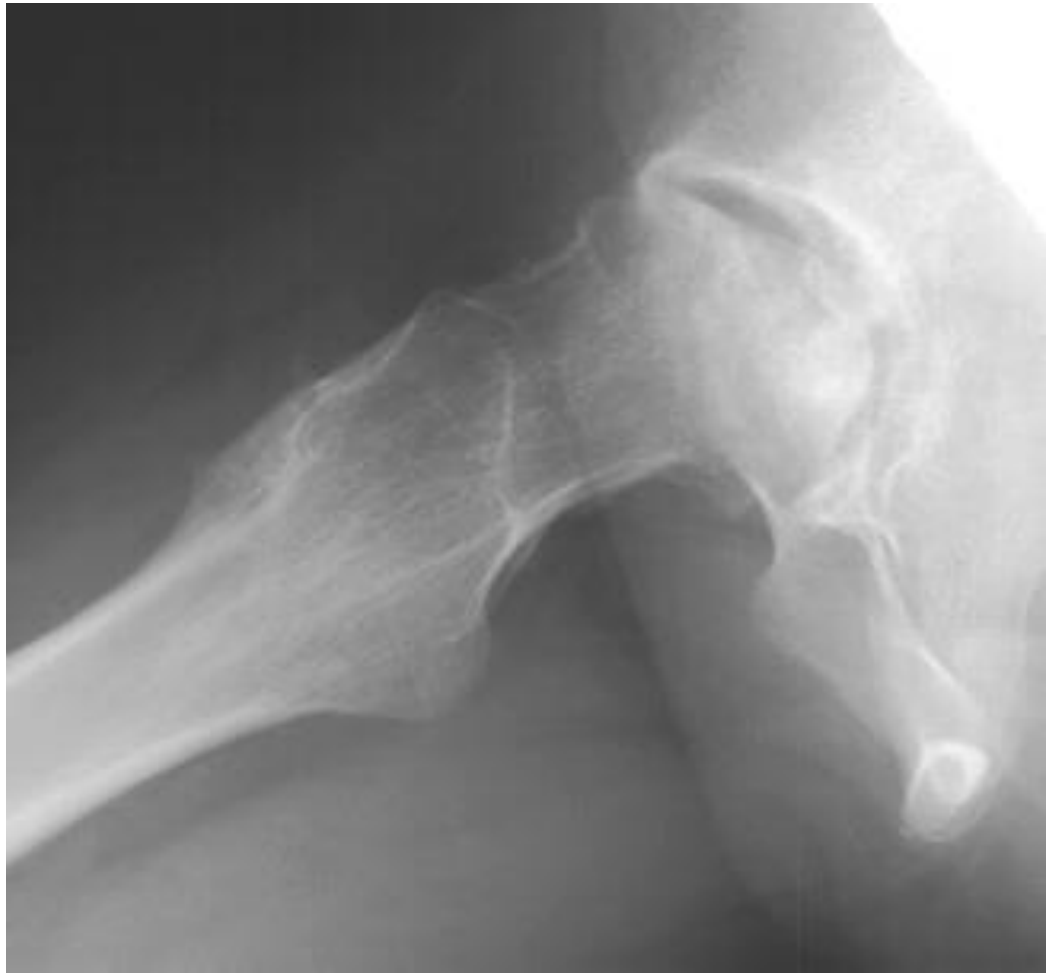
Closer image of spleen due to SCD

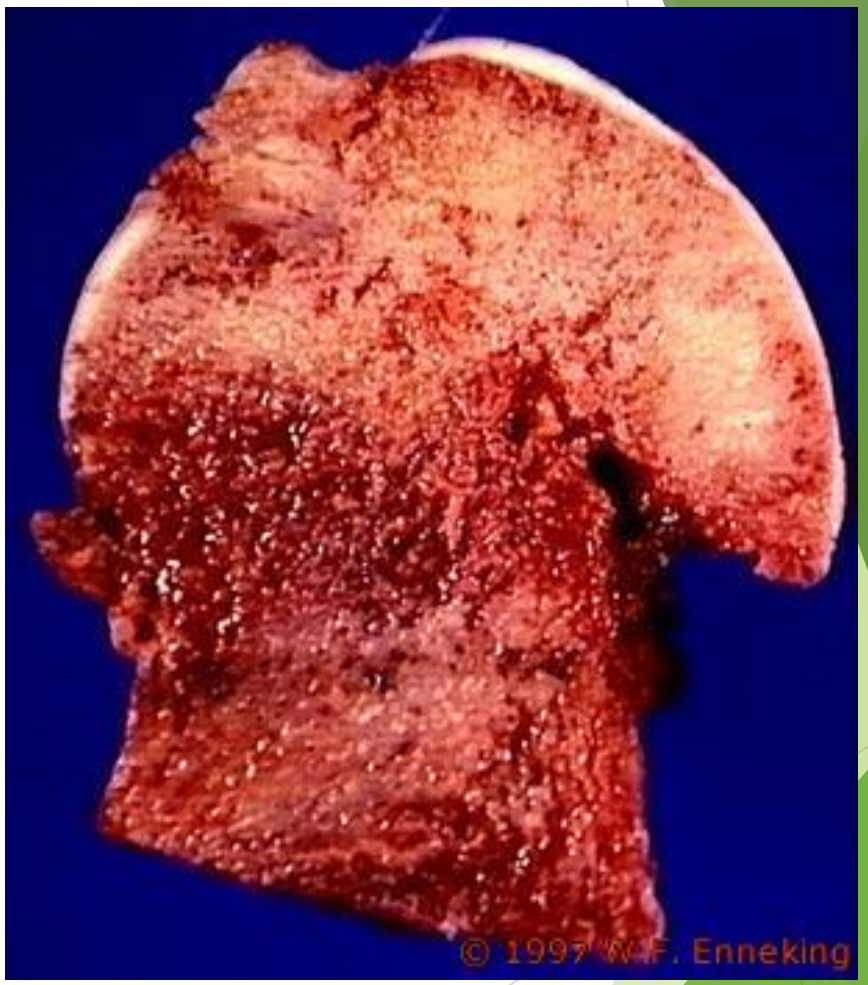


Auto-splenectomy - due to SCD

Figure 13-12: Splenic remnant in sickle cell anemia. (Courtesy of Drs. Dennis Burns and Darren Wirthwein, Department of Pathology, University of Texas Southwestern Medical School, Dallas, TX.)

Femoral head Necrosis due to SCD





Femoral head necrosis

Crew Cut

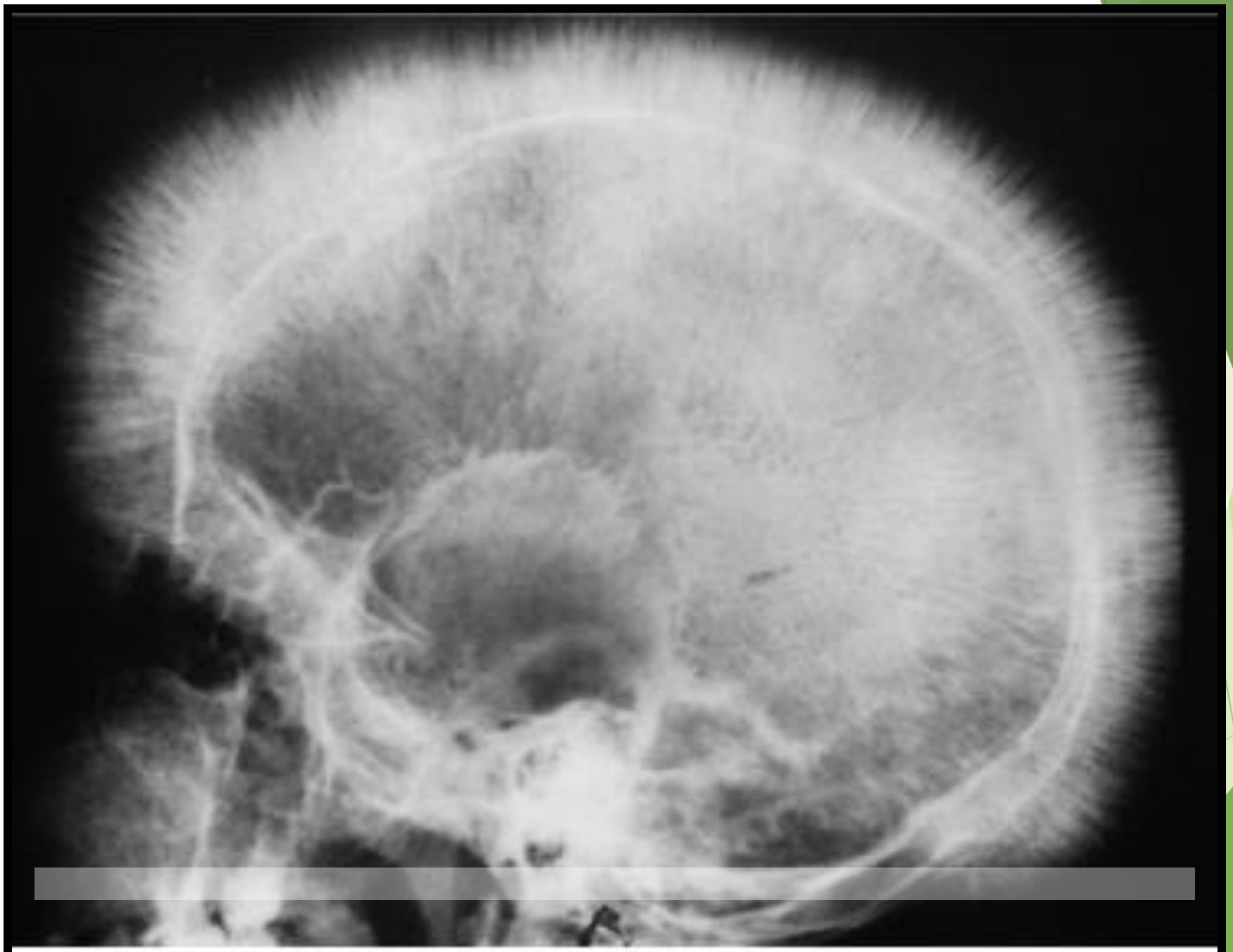
Calcification due to EMH

↓

also seen in Thalassemia

↓

skeletal deformities

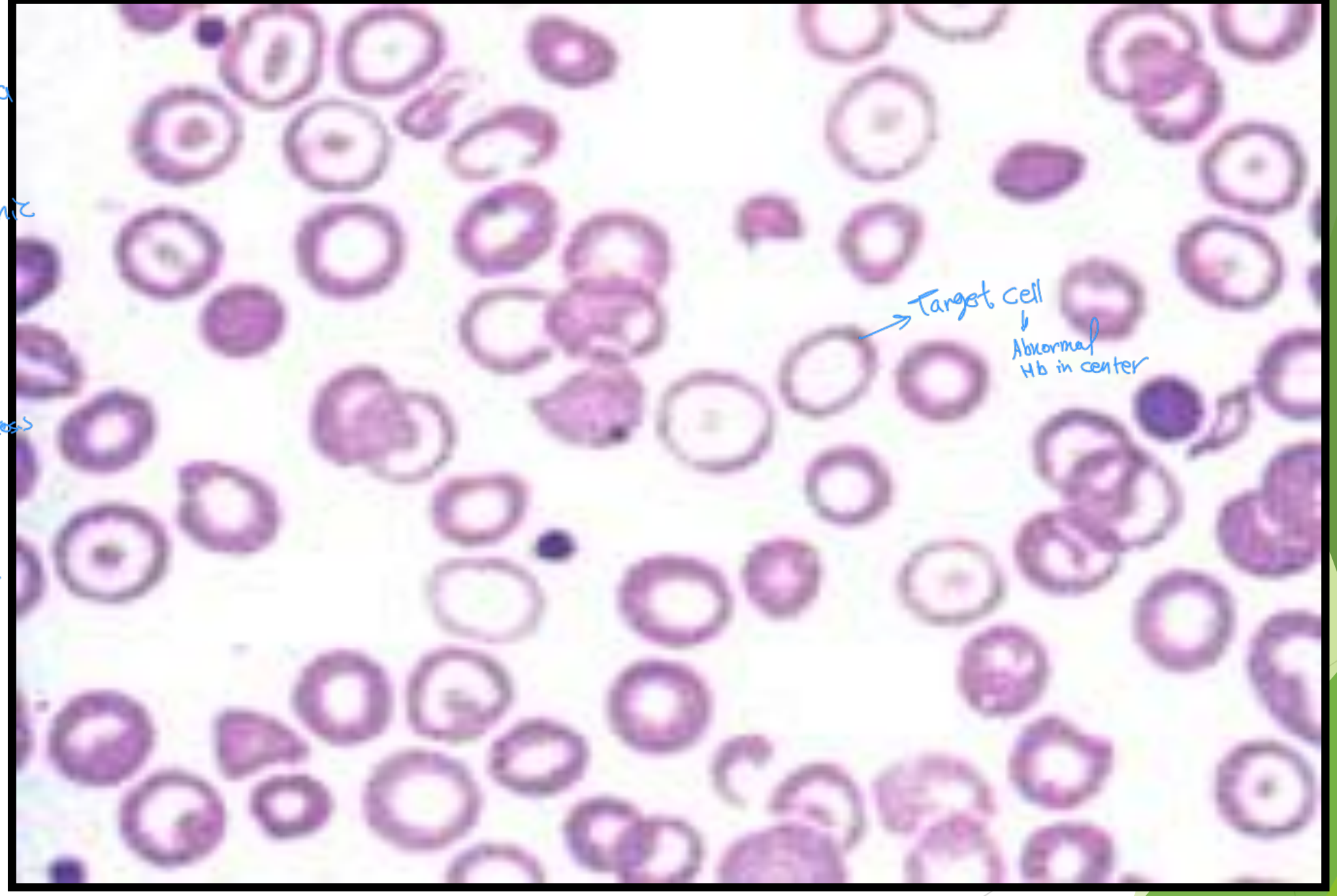


Thalassemia
↓
Microcytic
Hypochromic

- Cachexia
- Stunted growth
- 2° hemochromatosis
  ↓
  Cardiac dysfunction

α- Thalassemia
↓
Excess β → Hb H

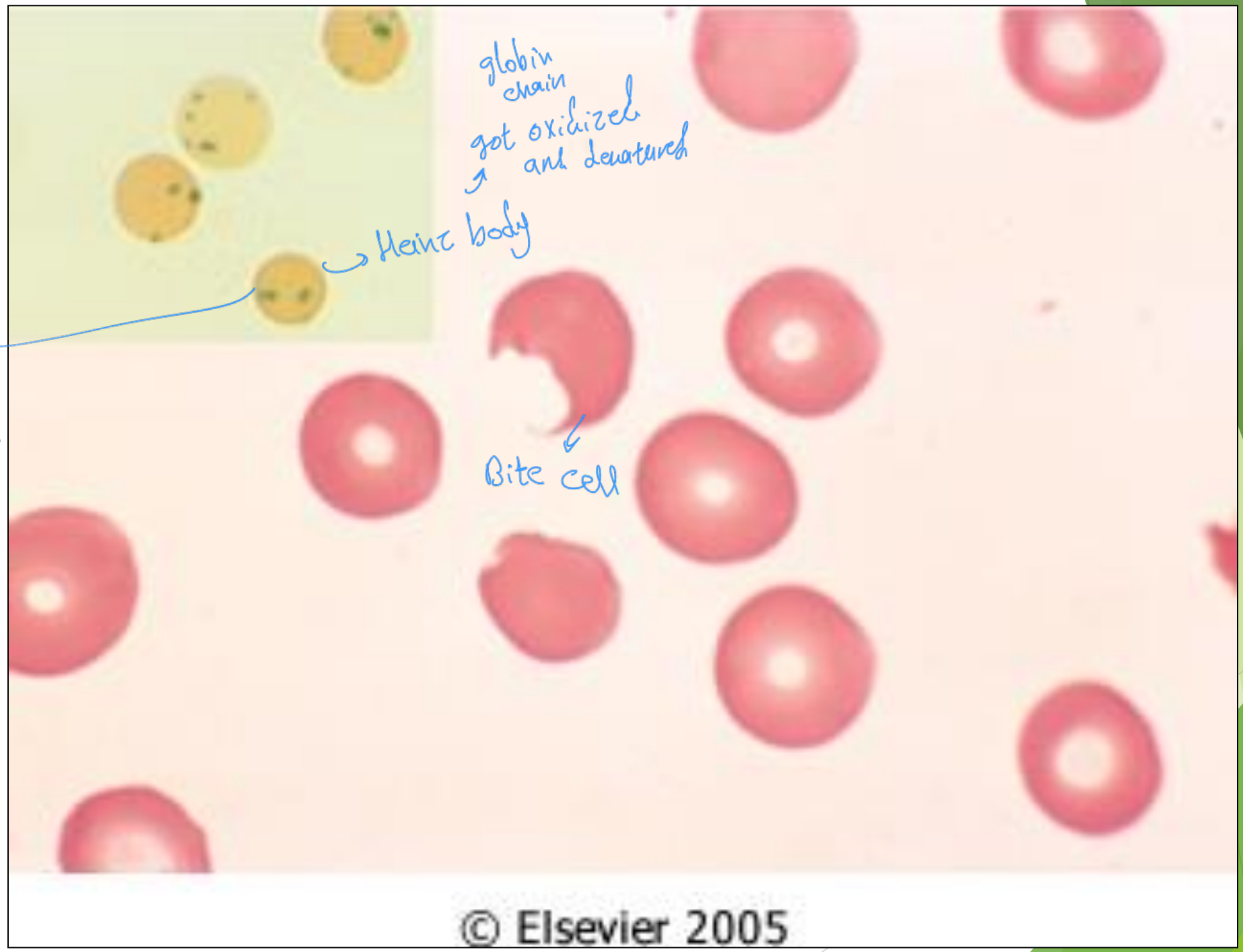
Excess γ → Hb barts
↳ Hydrops Fetalis

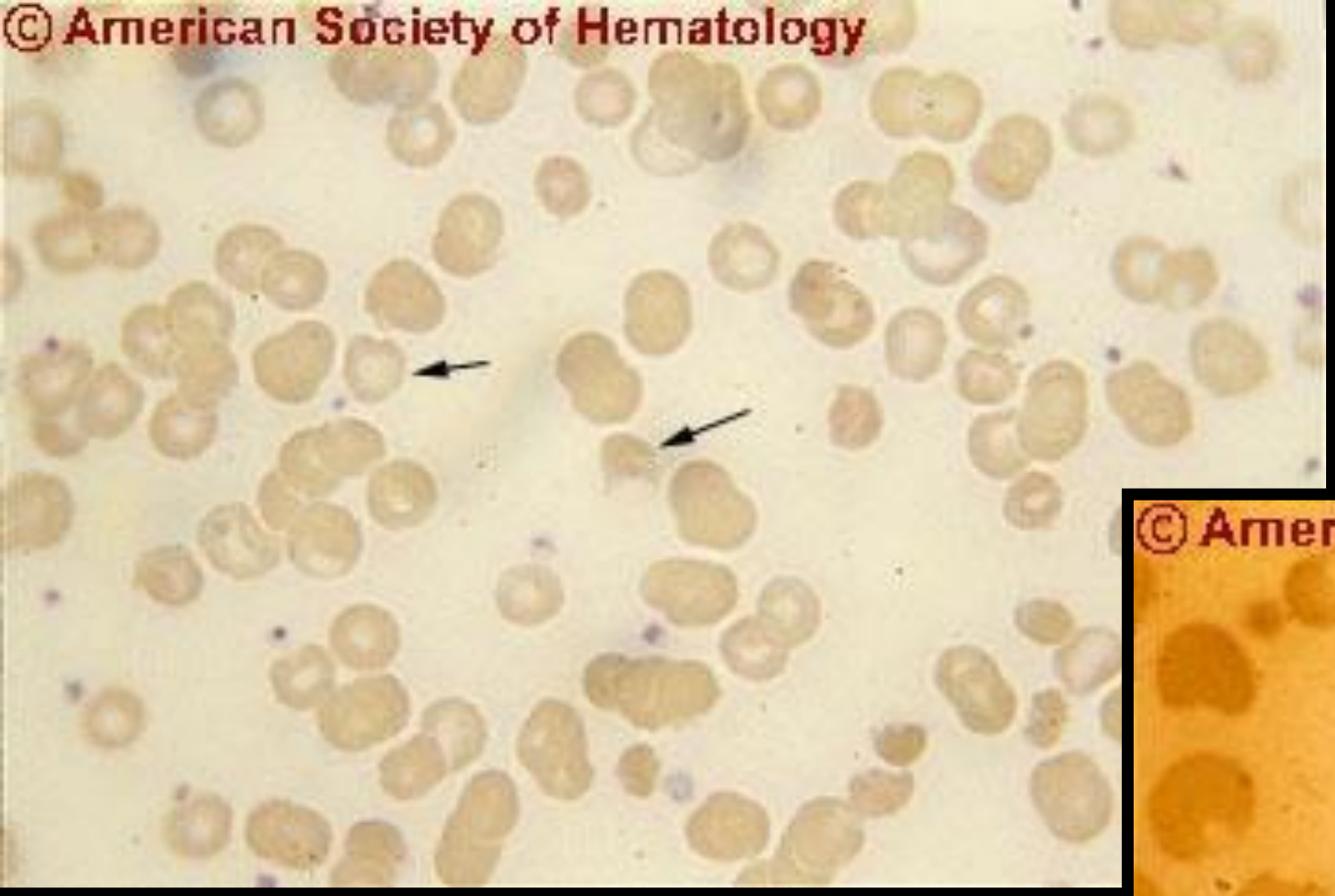


Hypochromic microcytic RBCs in Thalassemia

G6PD
x-linked

Also
Present
in Thalassemia

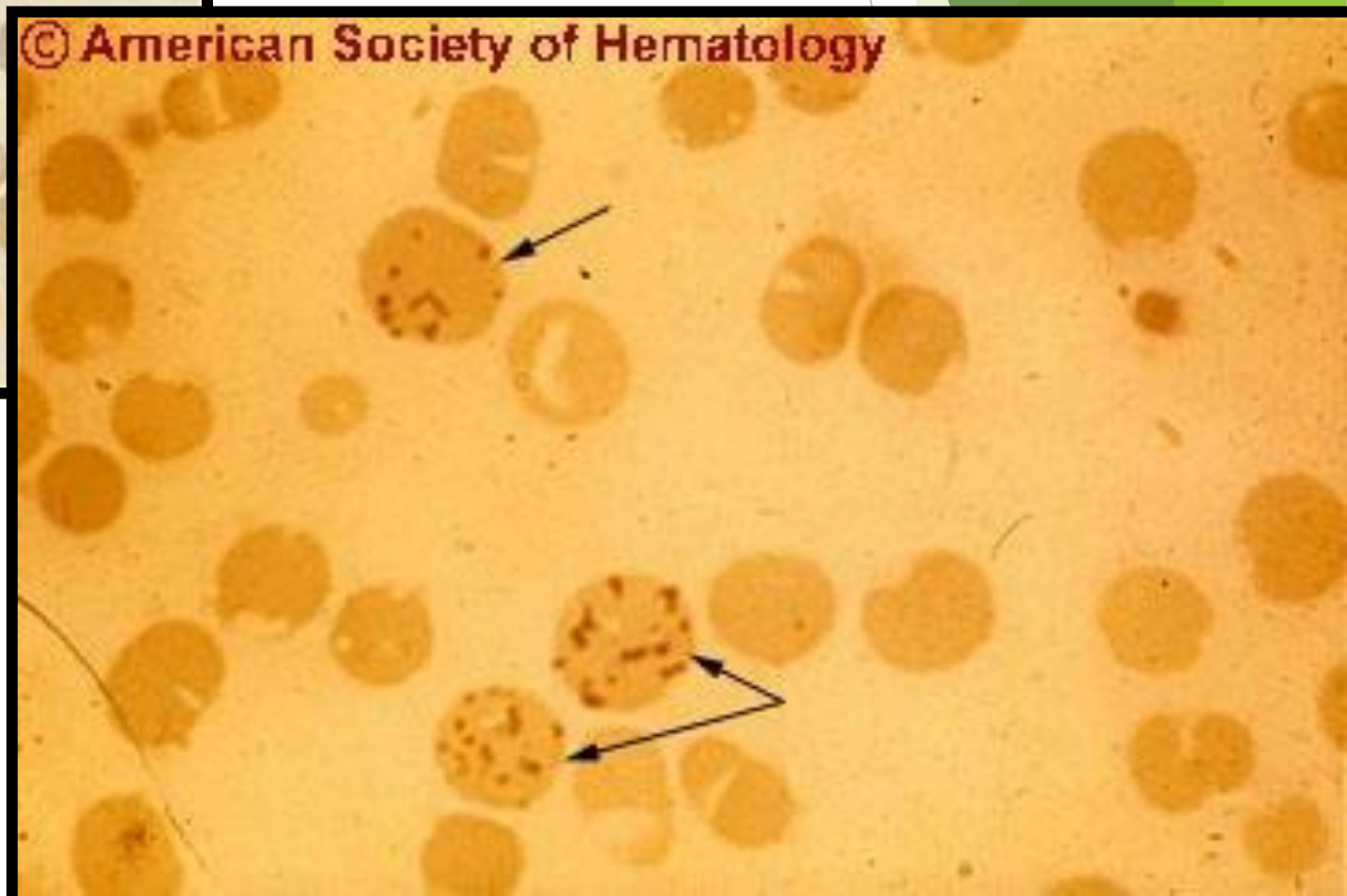




G6PD

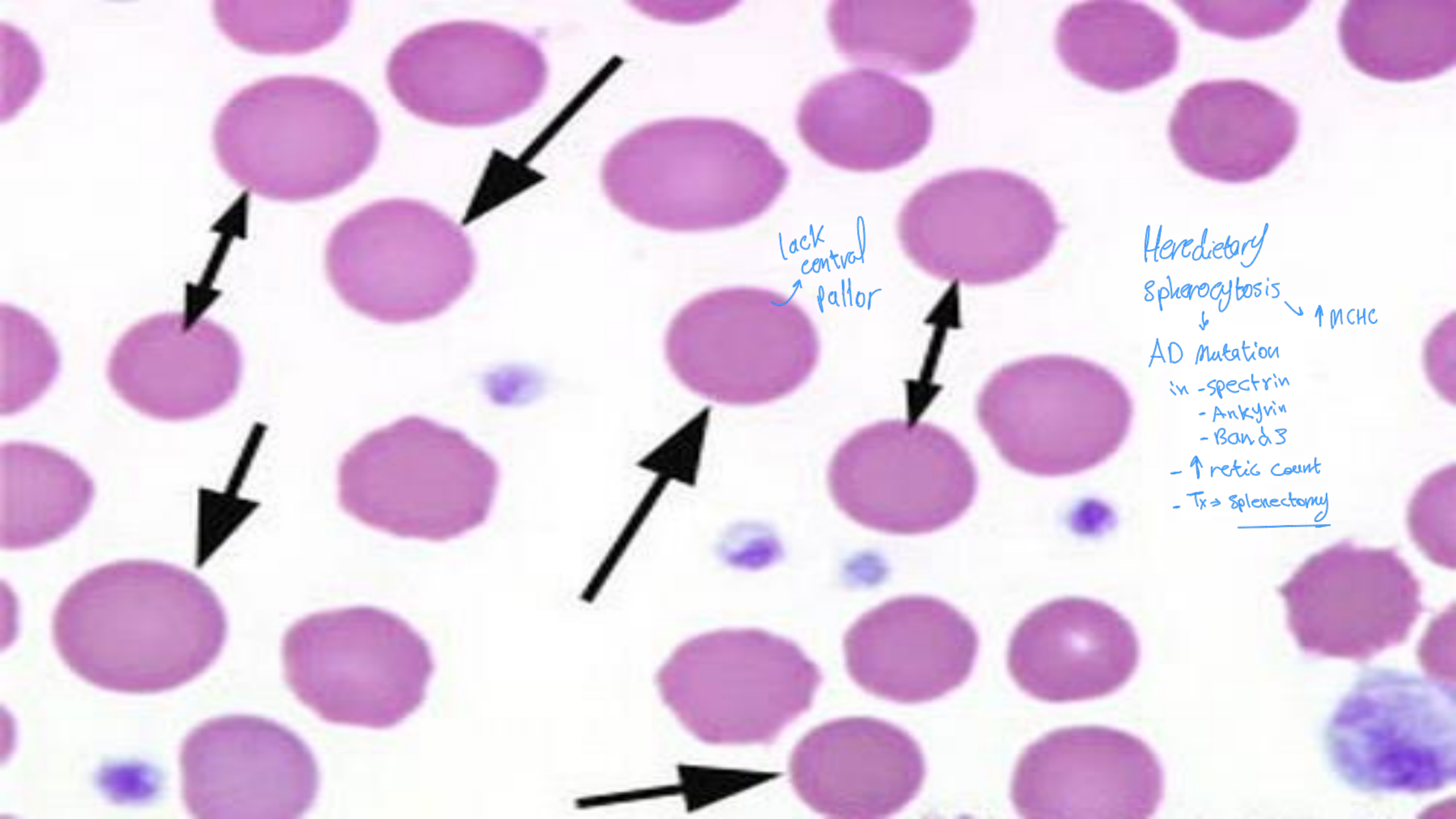
A-type → Age dependent

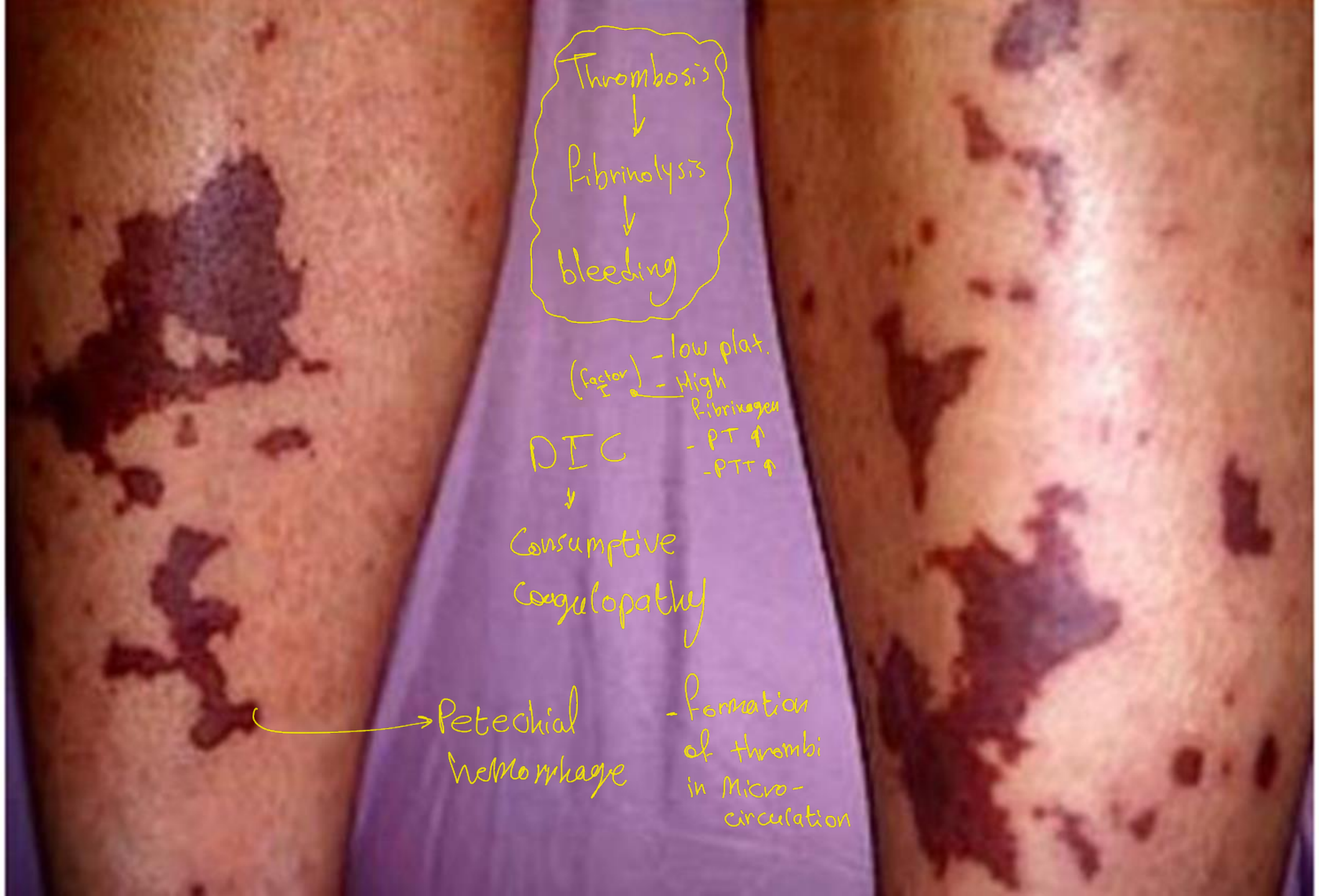
M-type → More severe
Age Independent



-Hemolytic Factorgs

- Favism
- Drugs → Anti-malarial, sulpha drugs
- Products of FR damage


lack central pallor
Hereditary Spherocytosis
↑ MCHC
AD mutation in -spectrin
- Ankyrin
- Band 3
- ↑ retic count
- Tx ⇒ splenectomy


Thrombosis
↓
Fibrinolysis
↓
bleeding
- low plat.
(factor I) - High fibrinogen
- PT ↑
- PTT ↑
DIC
↓
Consumptive coagulopathy
Petechial hemorrhage
- formation of thrombi in micro-circulation

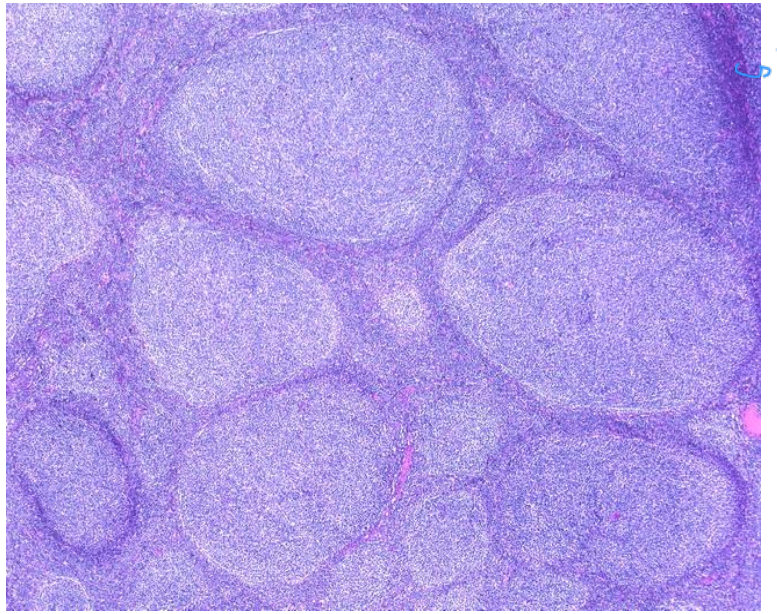
HLS-LAB

DR.EMAN KREISHAN

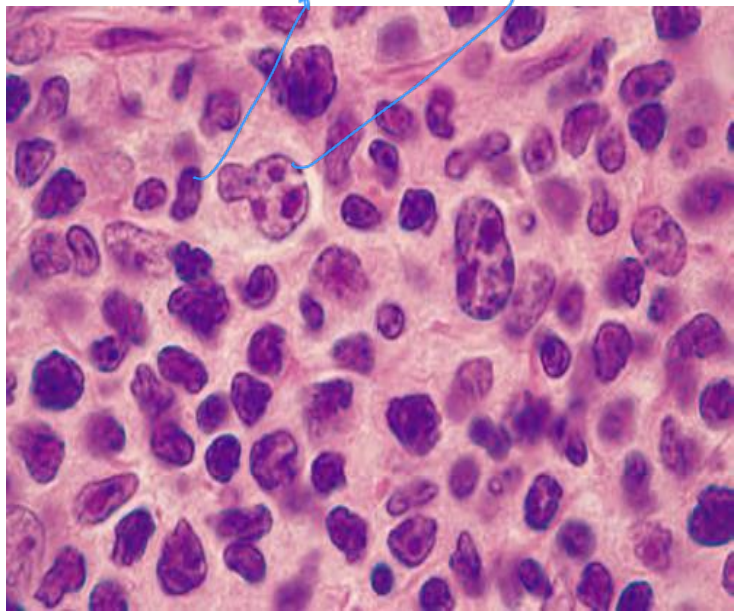
19-4-2023

Cancer	Cell Markers	Translocation /mutation	Histological feature
Follicular lymphoma	CD10 B cell markers (CD19,20,21)	14:18 ,BCL2	Increase no. Of follicles , centrocytes (small cleaved cells) + centroblast (large cleaved cells)
Mantle cell lymphoma	CD5 B cell markers (CD19,20,21)	11:14 cyclin D1	Increase no. Of follicles,but with irregular large lymphocyte
Marginal cell lymphoma	B cell markers (CD19,20,21)	11:18	Nodules and follicles with interfollicular lymphoid infiltration
Burkitt lymphoma	CD10 B cell markers (CD19,20,21)	8:14. c-myc	Starry sky appearance
Diffuse large B cell lymphoma	CD10 B cell markers (CD19,20,21)	BCL6	Diffuse large cells
Chronic lymphocytic leukemia /small lymphocytic lymphoma (CLL/SLL)	CD5 B cell markers (CD19,20,21) - TdT	PAX 5	Diffuse LN infiltration 1- (soccer ball cells) :large ,prominent nucleoli 2-small cells with dark round nuclei , clumped chromatin , scanty cytoplasm
Classic HL	CD15 , CD30 **No** B cell markers (CD20)		**Reed-sturnburg cells** + 1-nodules and fibrosis 2- background of mixed cellularity (eosinophils , lymphocytes ..etc) 3-back ground of only lymphocyte 4-background that is depleted from lymphocytes
Nodular lymphocyte predominant lymphoma	B cell markers (CD20) OCT-2 **No** CD15,CD30		**Popcorn cells** Vague nodules and fibrosis
T cell lymphoma	CD4+ , CD8-		Malignant T cell (cerebriform appearance) infiltrate dermis and epidermis

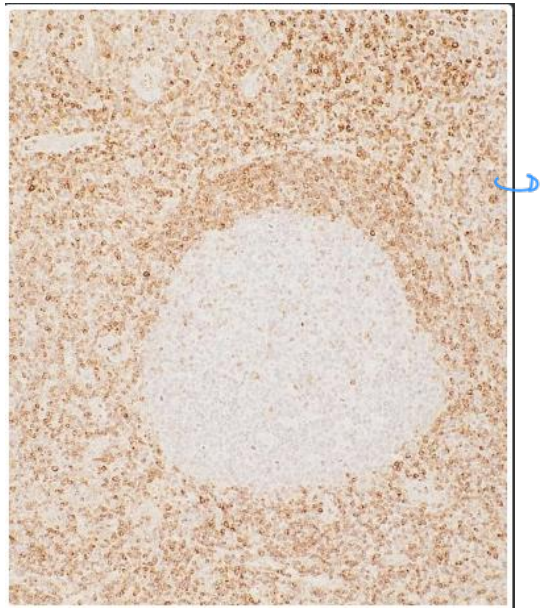
Follicular lymphoma. ↳ NHL → Nodular, Mature B-cell lymphoma



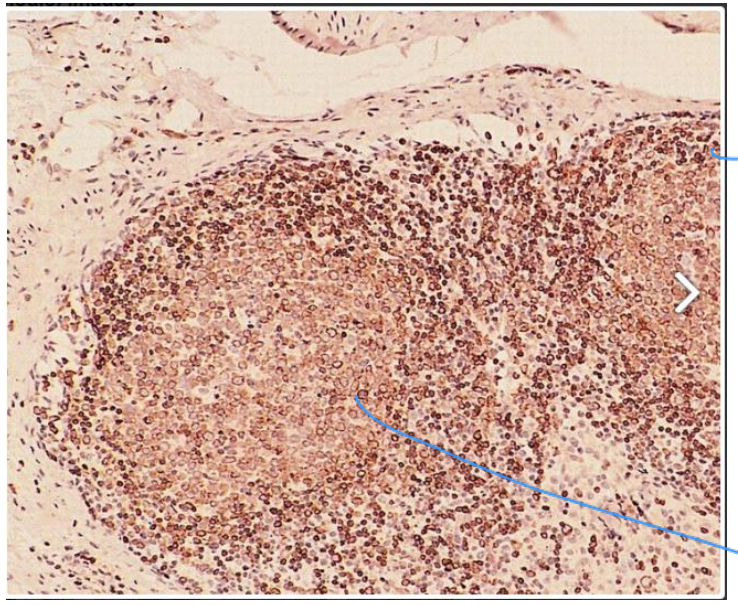
↳ Inter. Architecture of L.N are replaced by equally destribution between cortex and Medulla



↳ Higher Magnification



↳ CD10



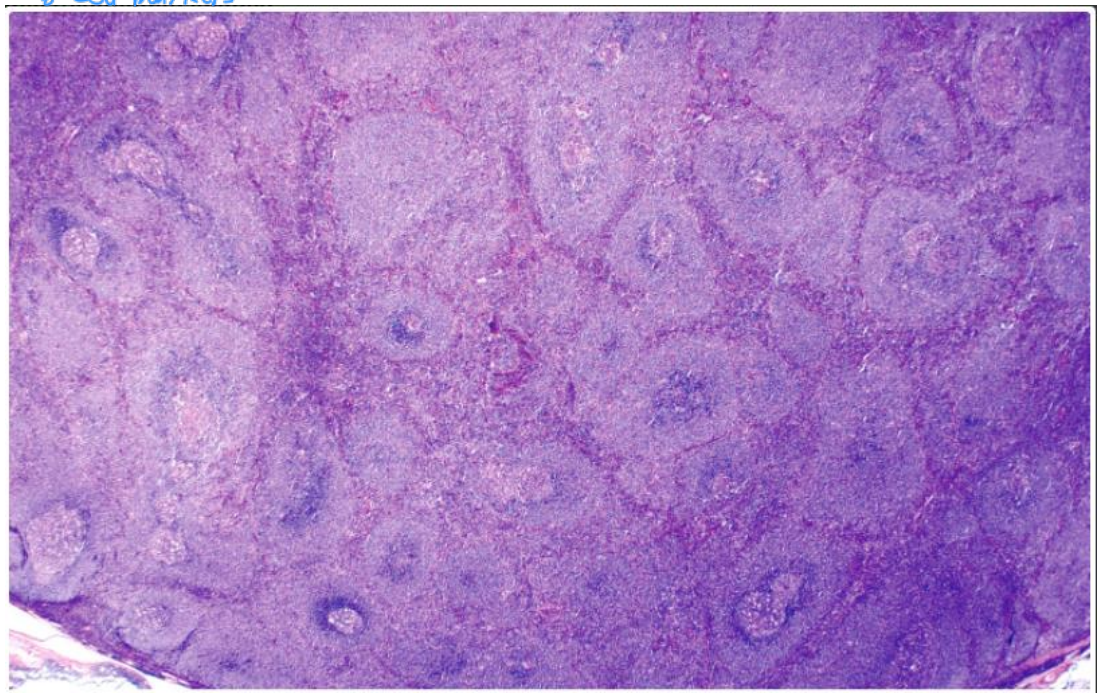
↳ BCL-2 Stain

-Ve in germinal centers
↓
Benign feature

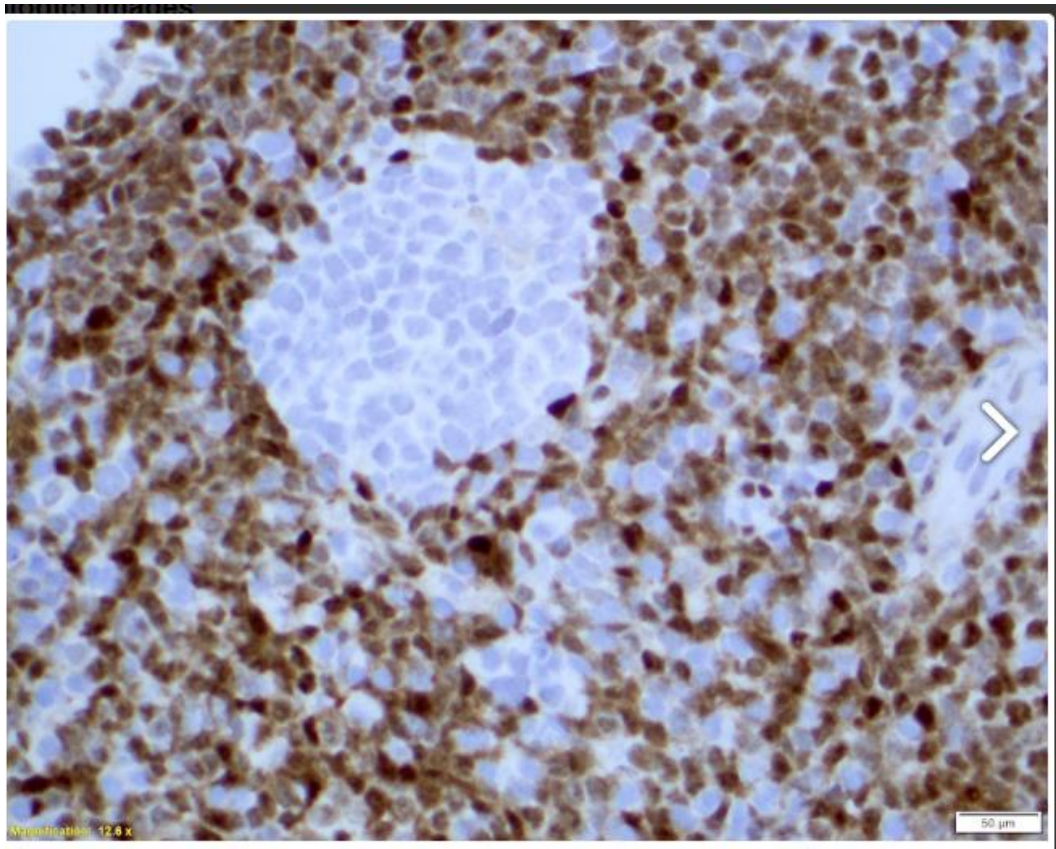
+Ve ↳ spot diagnosis of FL

Mantle Cell Lymphoma

- Multiple lymphoid follicels
- equall sizes
- same destribution
- B-cell markers

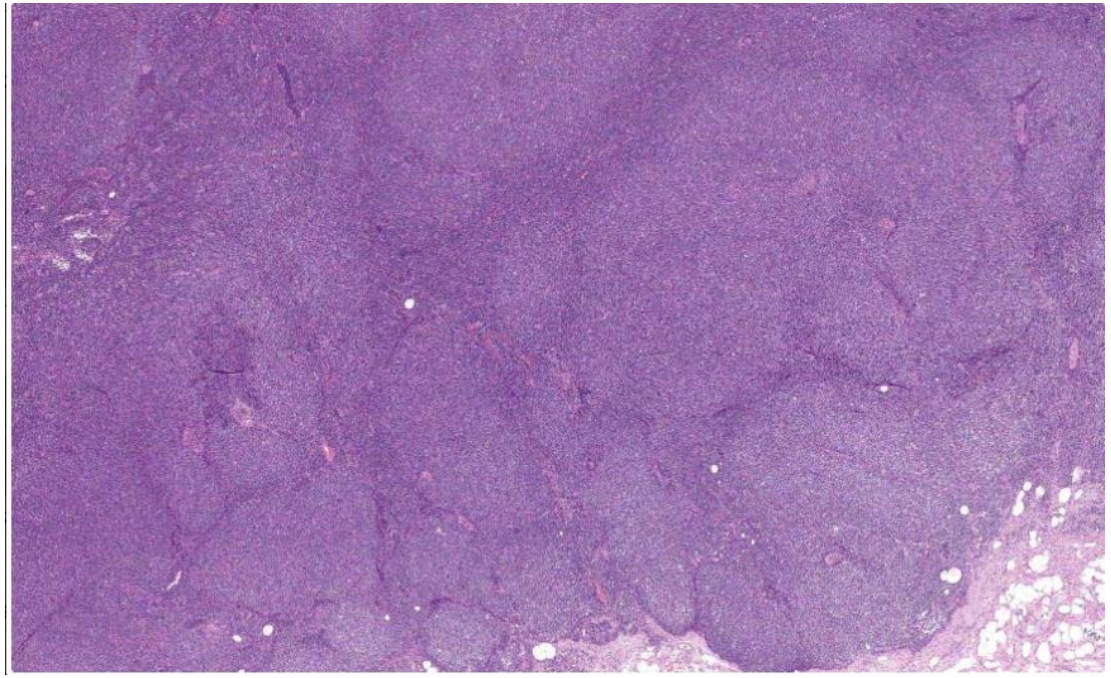


Cyclin-D1



Extranodal Marginal Zone Lymphoma

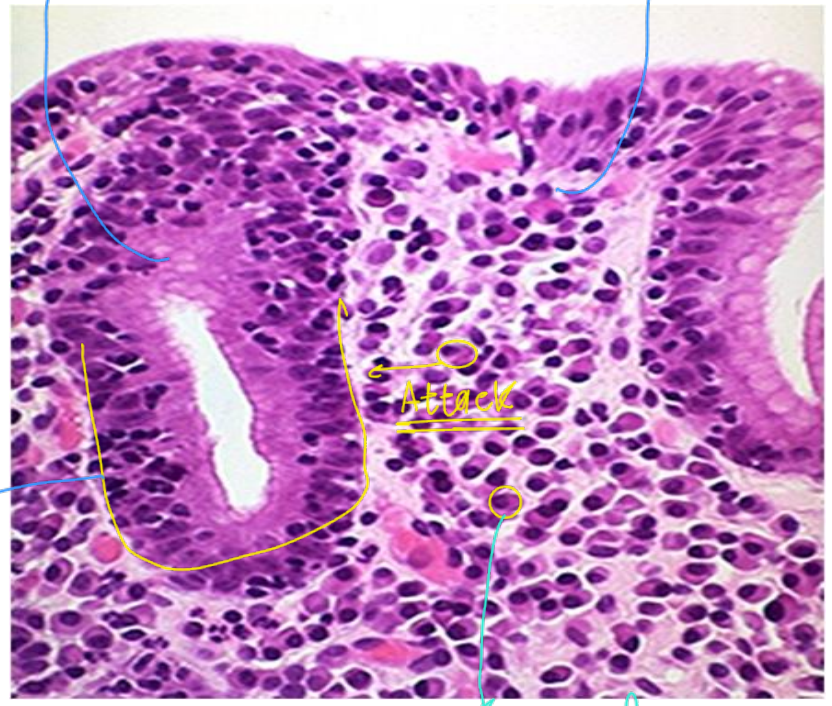
- Multiple lymphoid follicels



- gastric cites

MALT

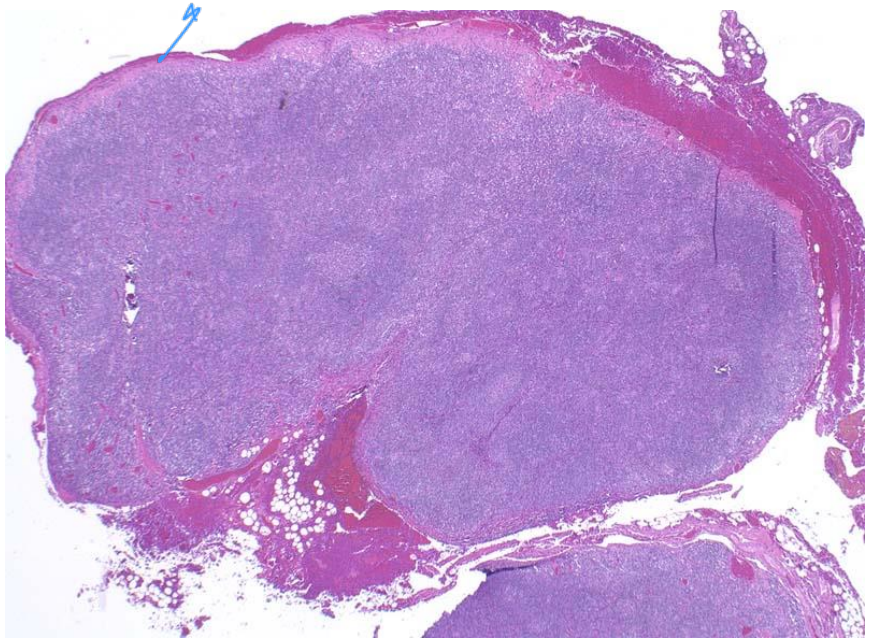
Lamina propria ↳ Contain Atypical lymphoid cells

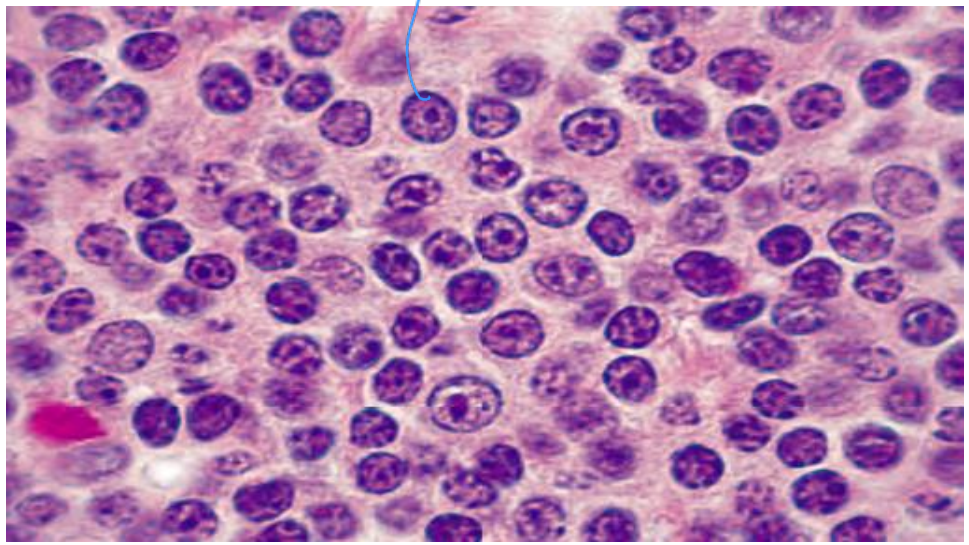


Diffuse

Chronic Lymphocytic Leukemia/Small Lymaphocytic Lymphoma (CLL/SLL)

- old Age

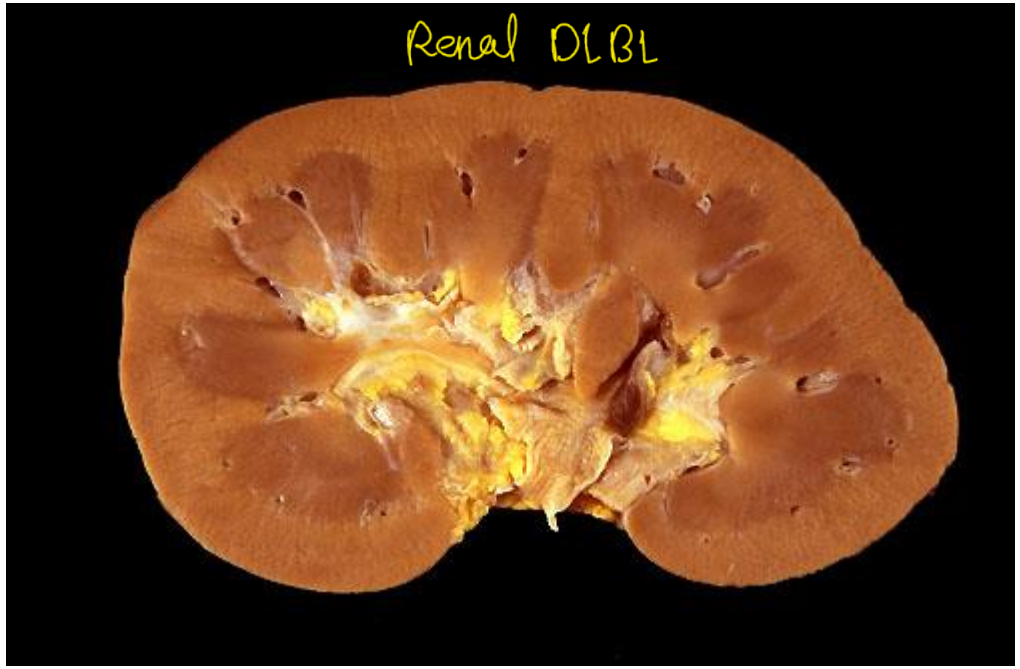


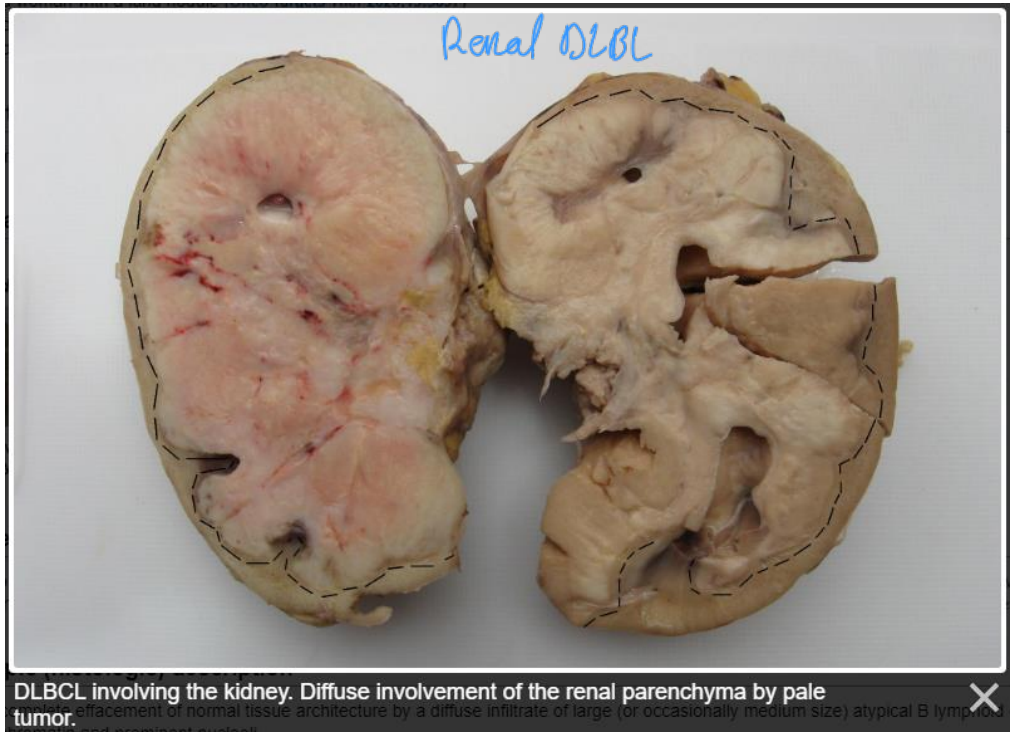


Diffuse Large B Cell Lymphoma

Grossly

- Very Aggressive
- Involve nodes + Extranodal sites

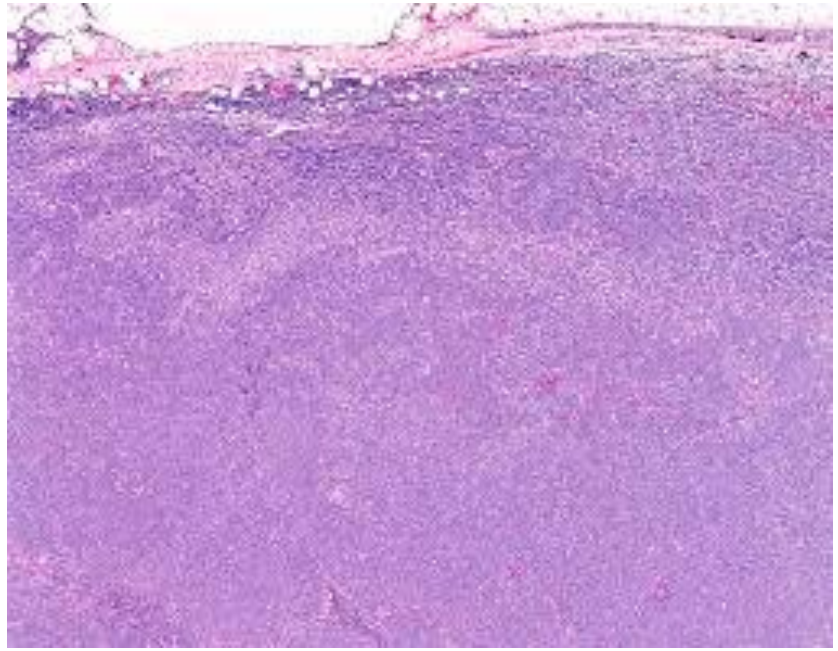




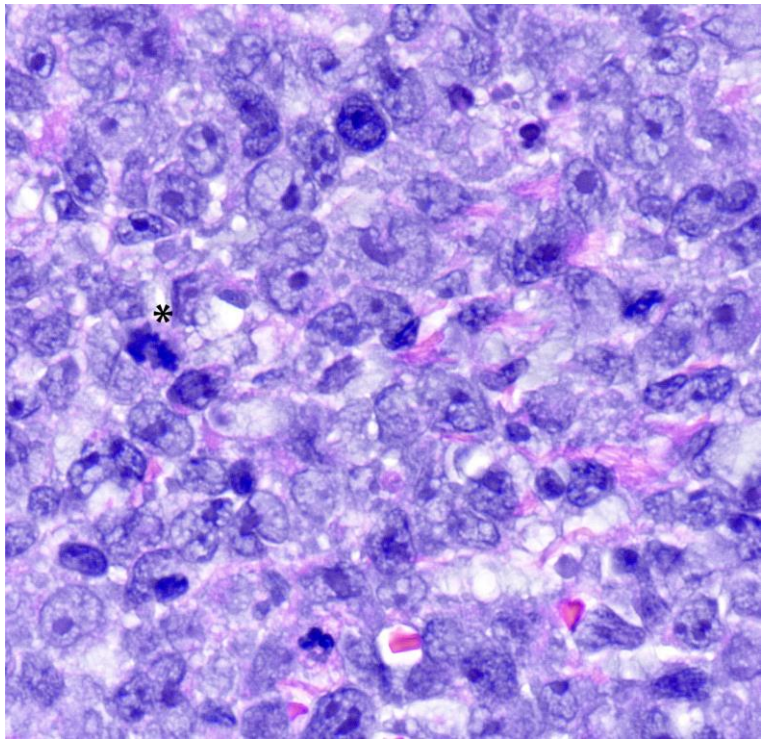
DLBCL involving the kidney. Diffuse involvement of the renal parenchyma by pale tumor.

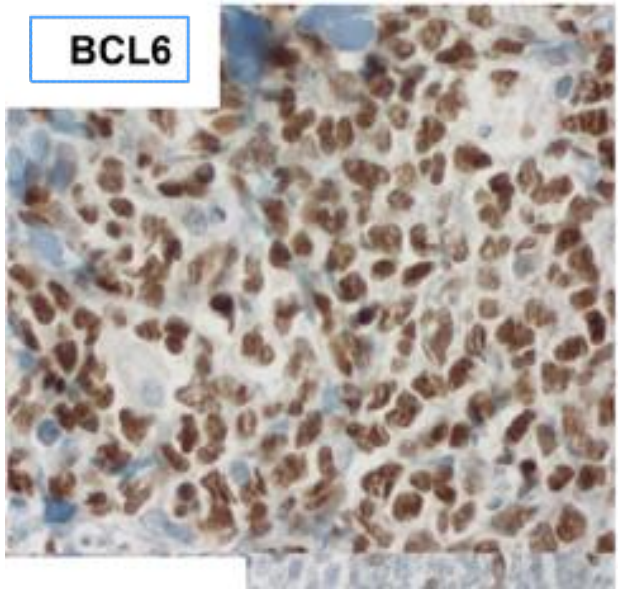
Diffuse Large B Cell Lymphoma

Diffuse sheet of lymphocytes



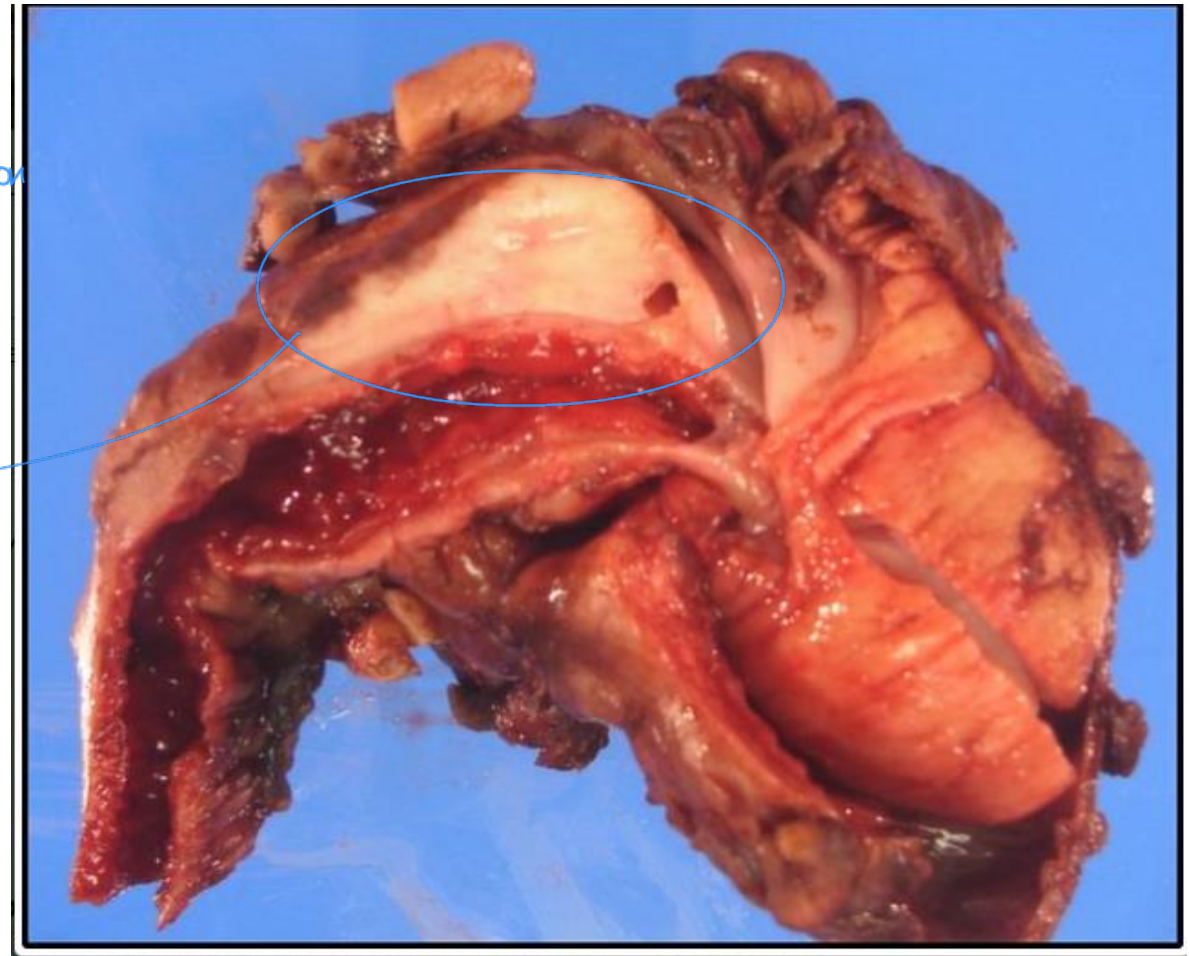
- Ugly cells
- 89 وكمة بتحب الثانية





Burkitt Lymphoma

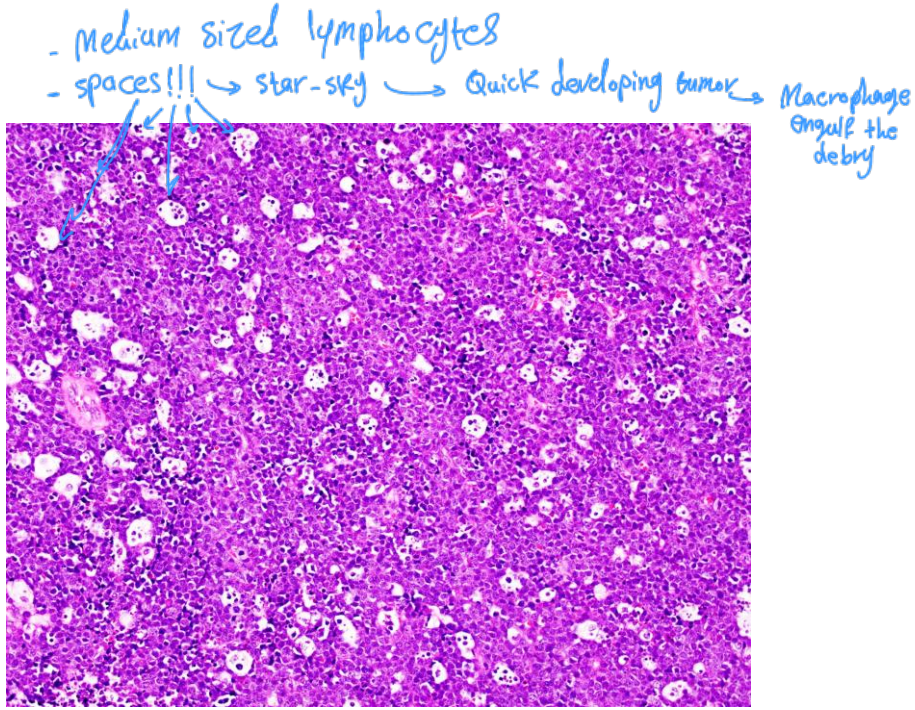
- children
- starry sky Appearance
- Myc mutation

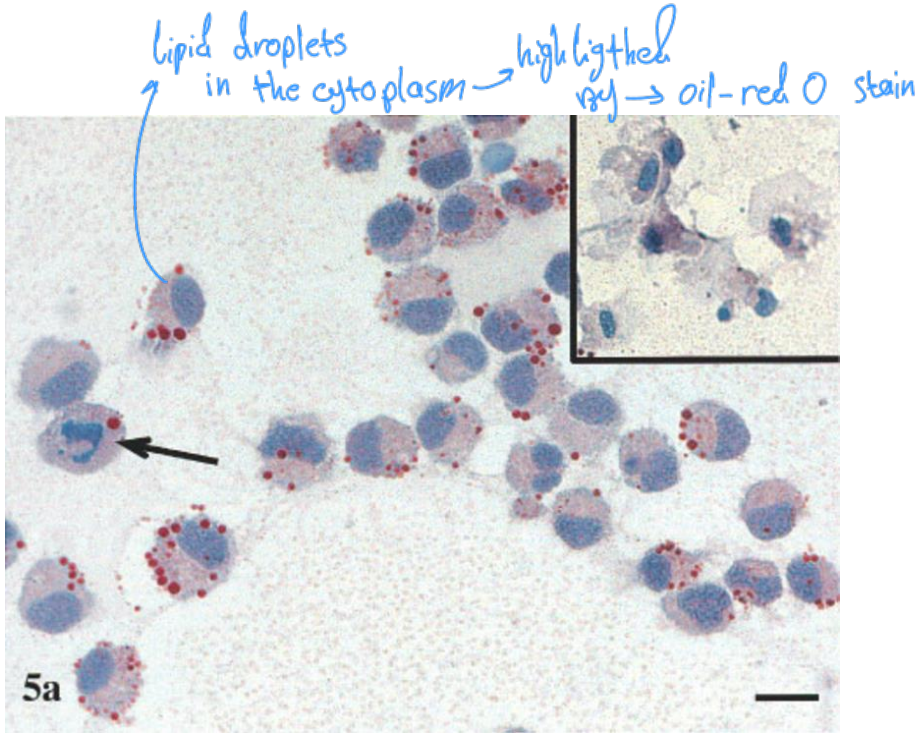


Fleshy homogenous mass invading the submucosa, consistent with Burkitt lymphoma.

Ileo-cecal Burkitt l.

Burkitt Lymphoma



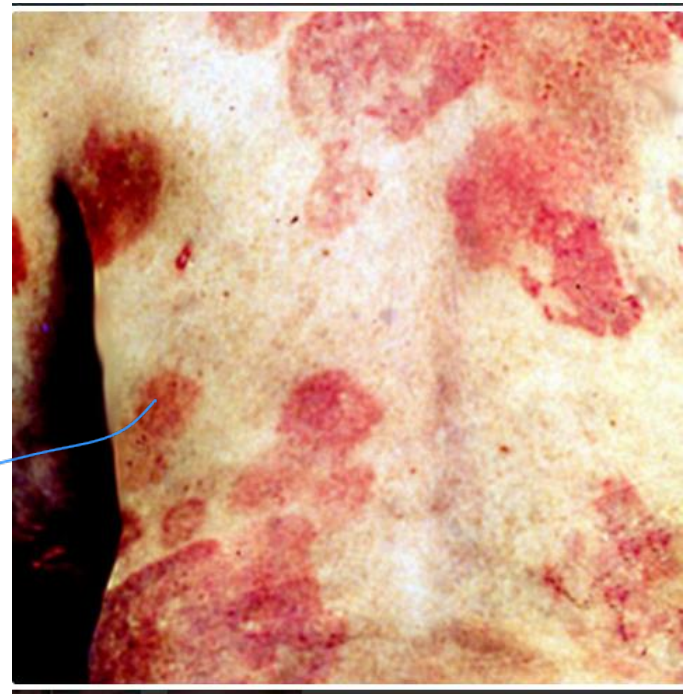


"MF"

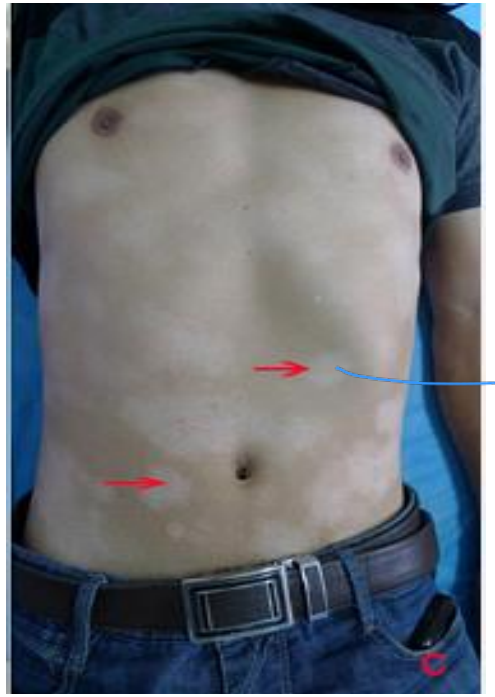
Mycosis Fungoides : T-cell lymphoma

Very Rare

- Very late Diagnosis → No Sus



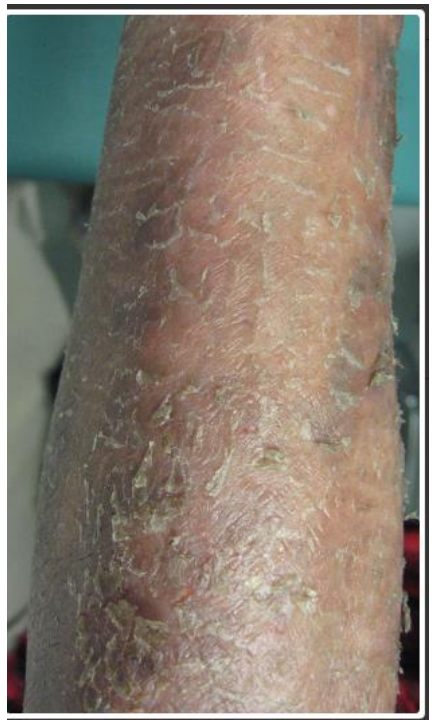
Rash
Adult

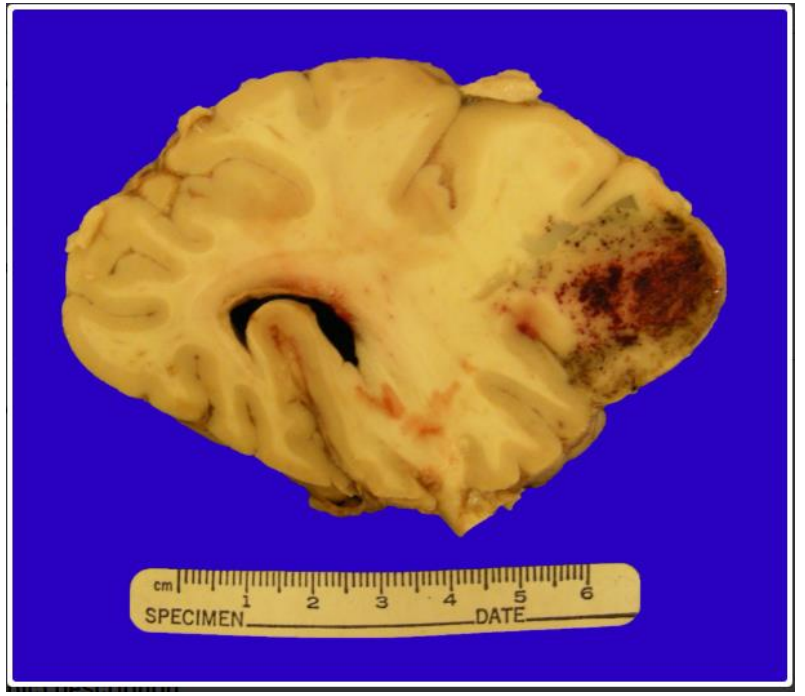


Hypo pigmentel lesions in Peds.

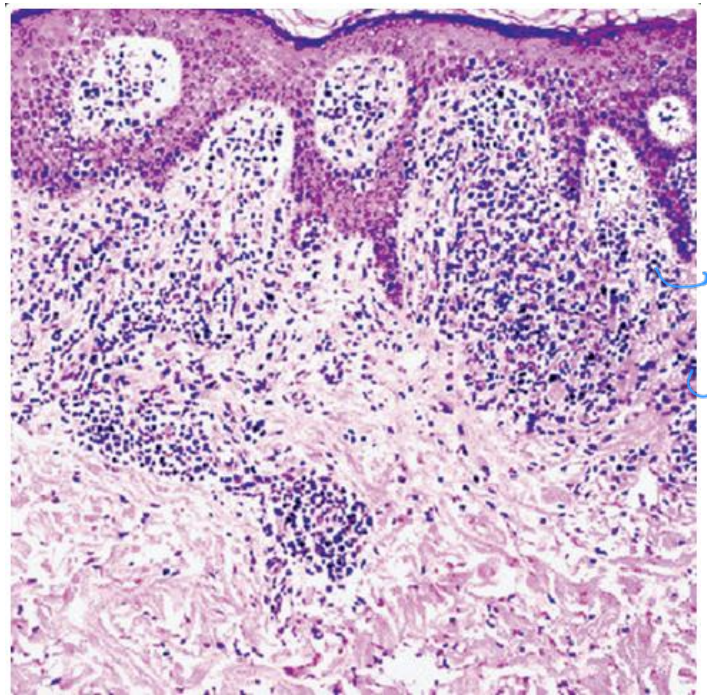
Sézary Syndrome:

if the neoplastic T-cells move from the skin → peripheral blood → organs



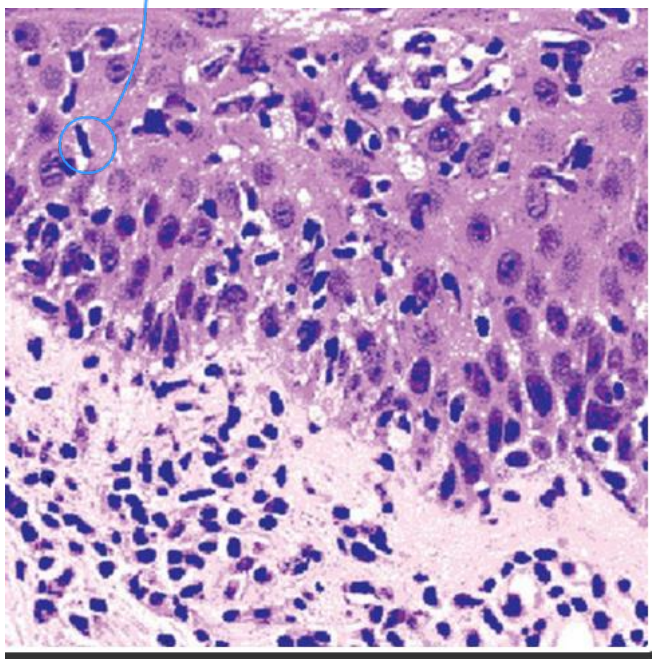


Mycosis Fungoides :



lymphoid cells

Heavy dermal + epi-dermal lymphocytic infiltrate.



Cerebroid lymphocytes → Elongated → Should be spherical.

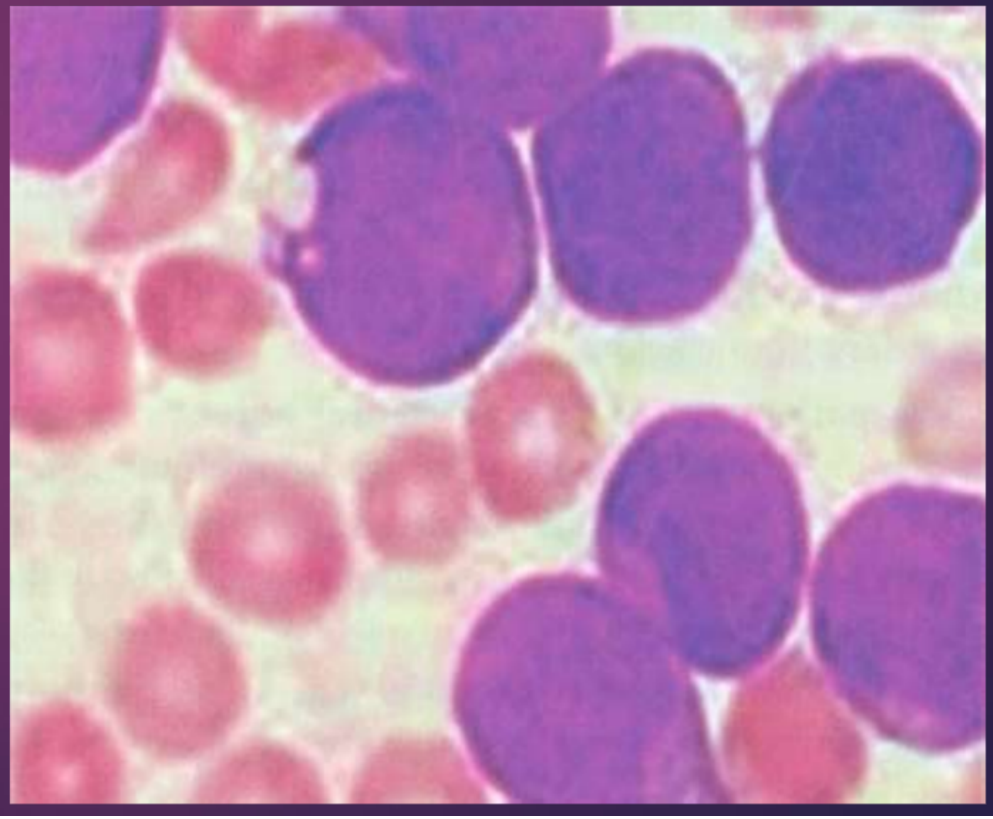
- L1 morphology: Lymphoblasts, are the most common subtype of childhood ALL (80-85%), have scant cytoplasm and inconspicuous nucleoli; these are associated with a better prognosis.
- Patients in the L2 category: accounting for 15% cases, show large, pleomorphic blasts with abundant cytoplasm and prominent nucleoli.
- Only 1-2% patients with ALL show L3 morphology: in which cells are large, have deep cytoplasmic basophilia and prominent vacuolation; these cells show surface imrnunoglobulin and should be treated as burkitt lymphoma.

ALL

Neoplastic lymphoblasts are slightly larger than lymphocytes and have scant, faintly basophilic cytoplasm and round or convoluted nuclei with inconspicuous nucleoli and fine chromatin, often in a smudged appearance

- Most common in children
- TdT +ve
- Associated w/ down syndrome (15-20)
- T-cell can present w/ mediastinal mass *
- May spread to CNS + testes
- translocation (12;21) or (8;14)

L1 Appearance

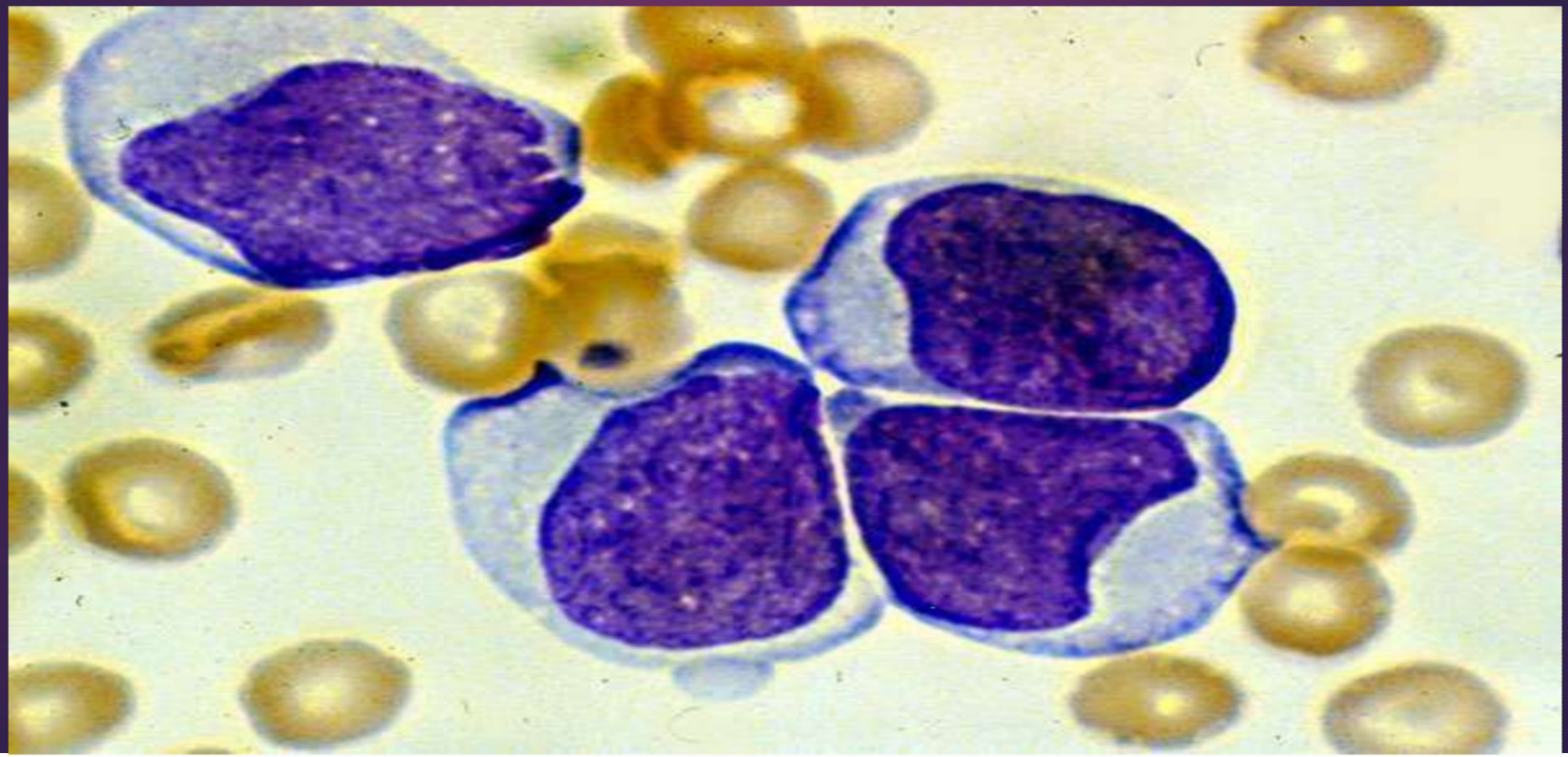


(*) Poor Prognosis Factors:-

1) Age <1 and >10
2) 9:22 t or 4:11 → infantile type
3) T-cell type w/ mediastinal mass
4) CNS or testicular spread
5) Initial WBC >20k
6) Poor response to initial chemo Tx

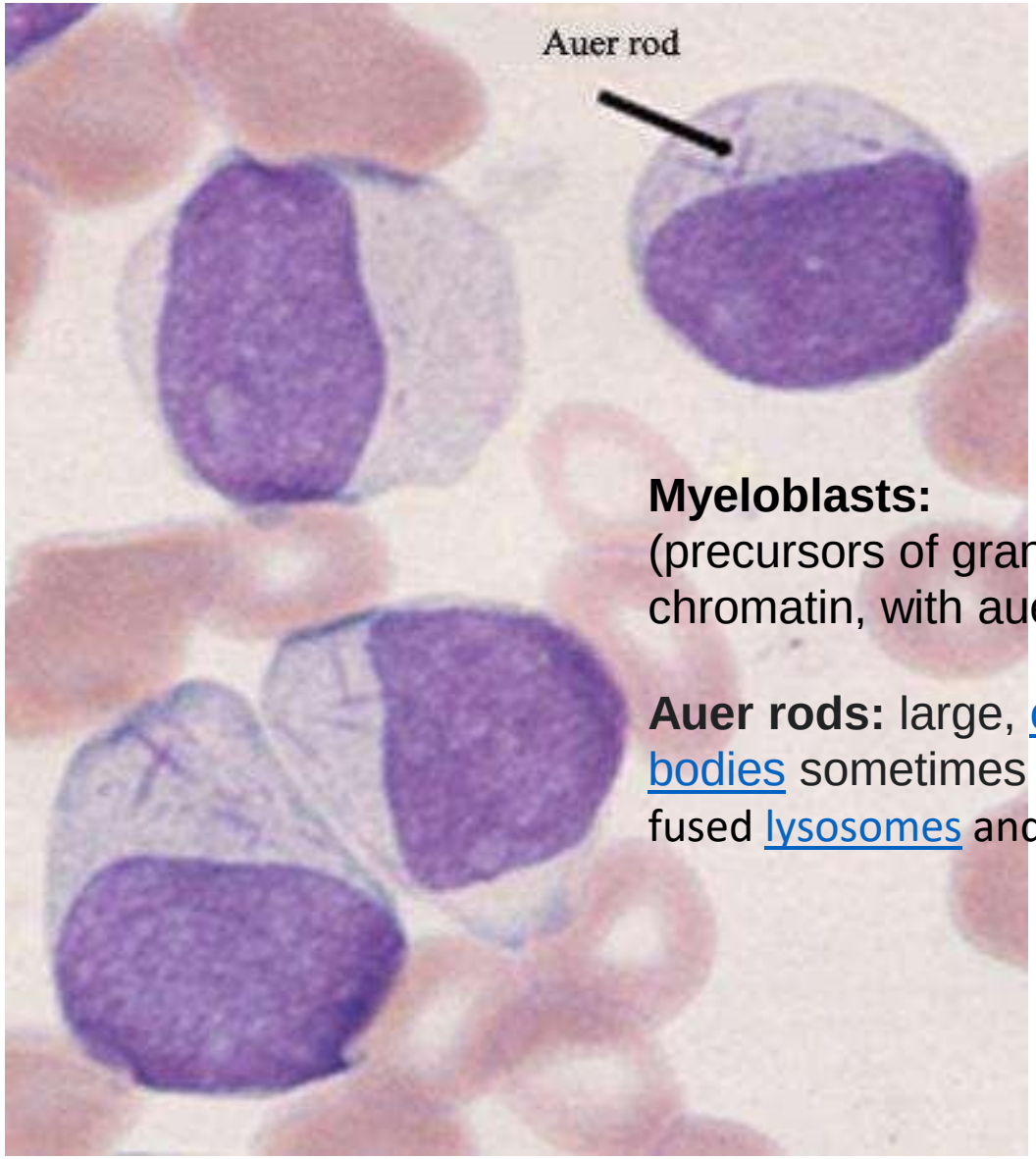
ALL

L2 Appearance



AML Histology

- Most often adolscence and after age 35y
- (15;17) translocation (PML;RARA)
- We use for treatment: ATRA + arsenic tri-oxide salt that degenerate the fusion protein



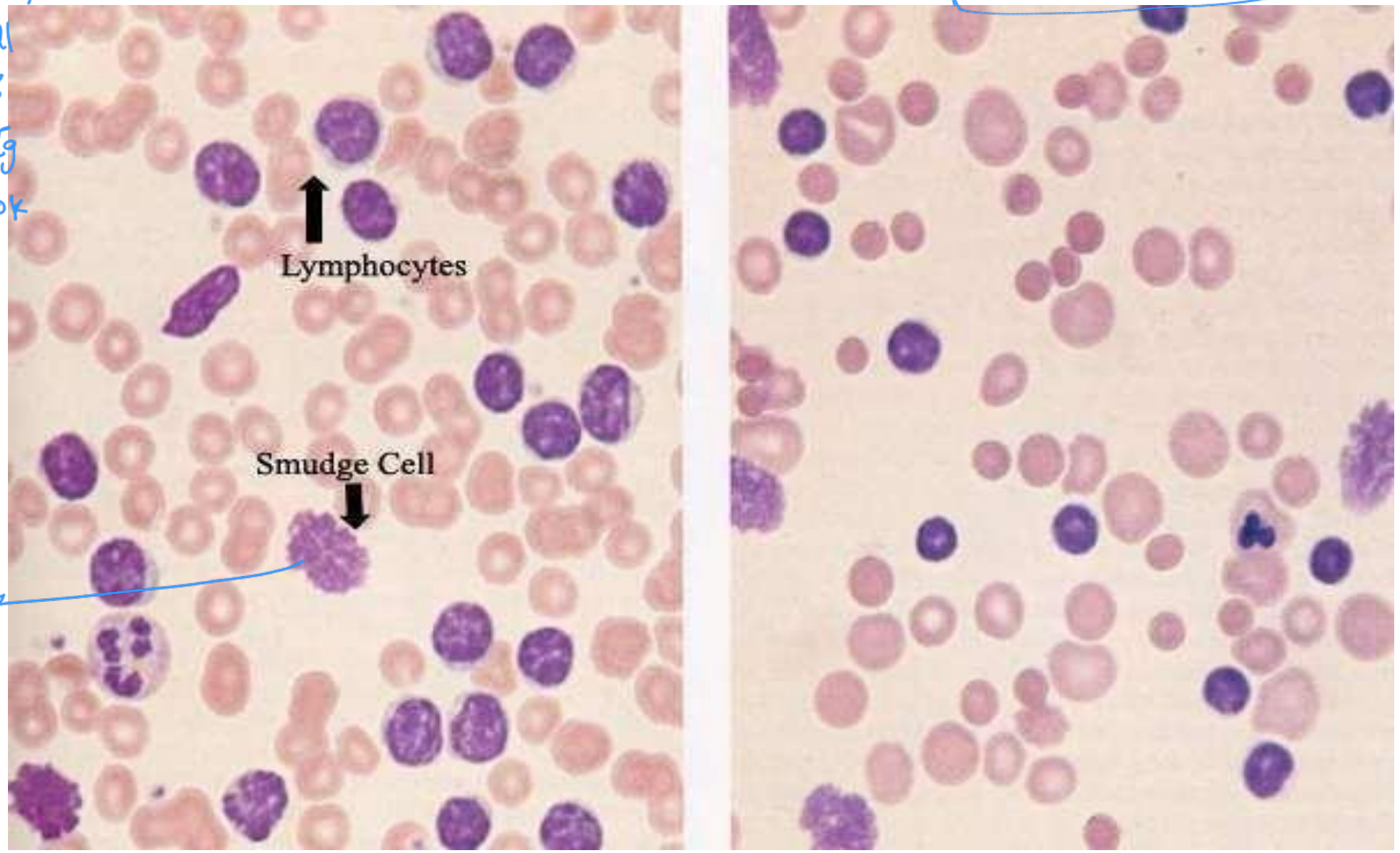
Myeloblasts:
(precursors of granulocytes) have delicate nuclear chromatin, with auer rods

Auer rods: large, crystalline cytoplasmic inclusion bodies sometimes observed in myeloid blast. Composed o fused lysosomes and rich in lysosomal enzymes

- Men, Age more than 60y
- Small, abnormal mature B-cell lymphocytes
- ↓ synthesis of Ig
- WBC 20k-100k

CLL Histology

we see smuged cells



Damaged cell

CML HISTOLOGY

- Caused by benzene exposure
  haigh dose of radiation
- 25 - 60 yrs.
- Peak 4th-5th decades of life
- Massive splenomegaly
- ↓ RBC, Hb, Hct
  early → ↑ platelet
  late → ↓ platelet
- WBC ↑ 100K
- Predominant Circulating granular cells (N, E, B)
- (9 ; 22) translocation
  (ABL; BCR)
  Philadelphia chromosome
  "good prognosis"